



ARCHIVES OF

*Physical Medicine
and
Rehabilitation*

APRIL 1961

Volume 42, No. 4

Archives of Physical Medicine and Rehabilitation

Code of Advertising

The *Archives of Physical Medicine and Rehabilitation*, published monthly by the American Congress of Physical Medicine and Rehabilitation, is interested in the maintenance of the highest standards in advertising in the interest of its readers in the medical and closely allied professions, the various manufacturers and patients. The *Archives of Physical Medicine and Rehabilitation* believes that honest, straightforward and informative promotion is essential in the merchandising of products and apparatus affecting the health and welfare of the general public. For this reason, the APM&R has formulated some basic principles to serve as a guide to manufacturers and advertisers.

CLAIMS

1. Claims for the efficacy or usefulness of a product should be logically and appropriately based upon scientific evidence or authoritative opinion expressed by qualified persons.
2. Claims should not be stronger than the evidence warrants and should be expressed in such a way as to avoid multiple interpretation. Implied endorsements by ambiguous statements are not acceptable. *The journal reserves the right to modify or exclude copy that is extravagant in claims without consulting advertiser and/or agent if time before publication deadline is a governing factor.*
3. Statements of endorsement or use of a product or apparatus by physicians or by institutions or by individuals in allied professions are not acceptable.
4. The statement of claims should be made in language appropriate to the understanding of the intended reading audience. If such statements are not clearly made, they may be misleading or confusing thereby doing disservice to both publisher and advertiser.
5. Length of time a product has been on the market and relative sales position are not necessarily evidence of its merit and hence such information cannot be considered appropriate argument for prescription or recommendation.
6. Promotion suggesting undesirable consequences from failure to use any product or apparatus unless compelling evidence exists is unjustifiable.

EVIDENCE

1. Evidence presented in promotion of products or apparatus should serve the function of validating any or all claims made. While interpretation of findings may vary among physicians, claims must adhere to the evidence and not to questionable extrapolations.
2. The significance of evidence should not be magnified to the point of overemphasis and quotations and/or excerpts should not disregard the context or the full meaning of the source.
3. Evidence should be readily and reasonably available. Manuscripts may be cited as references only after publication. Personal communication is acceptable only when the author has had an opportunity to review the intended promotion and has granted written approval for the citation. A copy of any personal communication to be included in advertising copy and a copy of permission directed to the manufacturer to use such personal communication must be on file in the office of the publisher prior to such information being released for publication in the journal. Descriptions of findings demonstrated in scientific exhibits not subsequently published in a generally available periodical do not constitute suitable evidence.
4. Evidence derived from adequately controlled research should be differentiated from uncontrolled clinical experience. Where the advertiser sets forth his own conclusion or rationale, it should be done without implication of other authority.

COMPARISONS

1. Promotion of a product or apparatus should relate the merits of the product or apparatus to generally acceptable medical standards.
2. Direct comparison with other products or apparatus for the purpose of demonstrating superiority is permissible only when such claim can be supported by direct reference to a published report.
3. Implied superiority of a product or apparatus through the overuse of superlatives is not acceptable.

POSITION OF THE MEDICAL PROFESSION

1. Promotion that tends to undermine the patient's trust and confidence in his physician is prejudicial to the effective practice of medicine and must be avoided.
 2. Promotion that results in the patient bringing pressure on the physician to prescribe according to suggestion or the current vogue rather than by exercise of professional judgment, is unacceptable.
 3. Promotion that encourages the patient to assume the prerogatives of the physician is unwise. It should be avoided as detrimental to public health. A prescription of drugs and/or therapeutic appliances leading to the time and sequence of the introduction of other treatment should remain the responsibility of the physician. Promotion influencing the patient to assume these responsibilities or creating concern regarding the physician's recommendations is not acceptable.
- The APM&R Code of Advertising conforms to the principle that promotion directly to the public of products and apparatus which require a physician's special knowledge for the proper administration is not in keeping with the best health and welfare of the general public. The APM&R Code of Advertising is subject to alteration and clarification as indicated by the experience and wisdom that will be acquired through cooperative efforts of medical, industrial, educational and public organizations.

ARCHIVES OF *Physical Medicine and Rehabilitation*

OFFICIAL JOURNAL AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION

★ AMERICAN ACADEMY OF PHYSICAL MEDICINE AND REHABILITATION

30 N. Michigan Avenue, Chicago 2, Illinois

Original contributions, exchanges and books for review should be forwarded to the Editorial Office. All business matters including advertising should be handled through the Executive Office, 30 N. Michigan Ave., Chicago 2, Illinois. Statements in the manuscripts published in the ARCHIVES OF PHYSICAL MEDICINE AND REHABILITATION are made solely on the responsibility of the author. The American Congress of Physical Medicine and Rehabilitation and/or the American Academy of Physical Medicine and Rehabilitation does not assume any responsibility for statements contained therein. Manuscripts accepted for publication in the ARCHIVES OF PHYSICAL MEDICINE AND REHABILITATION are for exclusive publication and may not be published elsewhere.

Subscription—In the United States, its possessions, and Mexico, \$8.50 yearly; Canada, \$9.50; elsewhere, \$14.00 the year; \$1.00 per single copy; special issues \$1.50 per single copy. Advertising rates on application. All advertising copy subject to acceptance by Publication Committee. Permission will be granted on request for reproduction in reputable publications if proper credit is given. Reproduction for commercial purposes will not be permitted. Published monthly at Chicago, Illinois, by the American Congress of Physical Medicine and Rehabilitation. Entered as Second Class Matter at the Post Office at Chicago, Illinois. Copyright 1961, by the American Congress of Physical Medicine and Rehabilitation.

American Congress of Physical Medicine and Rehabilitation OFFICERS

DONALD A. COVALT
New York, President
DONALD J. ERICKSON
Rochester, Minn., President-Elect
JEROME S. TOBIS
New York, First Vice-President
LOUIS B. NEWMAN
Chicago, Second Vice-President
CHARLES D. SHIELDS
Washington, D. C., Third Vice-President
WILLIAM J. ERDMAN, II
Philadelphia, Fourth Vice-President
LEWIS A. LEAVITT
Houston, Fifth Vice-President
JOSEPH G. BENTON
New York, Secretary
FRANK H. KRUSEN
Rochester, Minn., Treasurer
GLENN GULLICKSON, JR.
Minneapolis, Executive Director
DOROTHEA C. AUGUSTIN
Chicago, Executive Secretary

EXECUTIVE COUNCIL

Frederic J. Kottke, Minneapolis, *Chairman*.
Norman E. Tuttle, Verona, N. J., *Secretary*.
Robert L. Bennett, Warm Springs, Ga.
William Bierman, San Francisco.
Earl C. Elkins, Rochester, Minn.
Kristian G. Hansson, New York.
Abraham R. Hollender, Miami Beach, Fla.
O. Leonard Huddleston, Santa Monica, Calif.
Arthur C. Jones, Portland, Ore.
H. Worley Kendal, Peoria, Ill.
Miland E. Knapp, Minneapolis.
A.B.C. Knudson, Washington, D. C.
Frank H. Krusen, Rochester, Minn.
Gordon M. Martin, Rochester, Minn.
Walter S. McClellan, Chapel Hill, N. C.
Fred B. Moor, Los Angeles.
William D. Paul, Iowa City.
Nathan H. Palmer, New Orleans.
Donald L. Rose, Kansas City, Kans.
William H. Schmidt, Philadelphia.
Wm. Benham Snow, New York.
Arthur L. Watkins, Boston.
Donald A. Covalt, New York, *Ex-officio*.

PRINTED IN U.S.A.

EDITORIAL BOARD

PAUL A. NELSON, Cleveland, *Chairman*
ARTHUR C. JONES, Portland, Ore.
FREDERIC J. KOTTKE, Minneapolis
OSCAR O. SELKE, JR., Houston
G. KEITH STILLWELL, Rochester, Minn.
JEROME S. TOBIS, New York
ARTHUR L. WATKINS, Boston

The ARCHIVES OF PHYSICAL MEDICINE AND REHABILITATION is devoted to the publication of significant articles in this particular field. Contributions of interest to those in physical medicine and rehabilitation are invited. Articles are accepted only with the understanding that they are contributed exclusively to the ARCHIVES. Articles and accompanying illustrations become the property of the journal.

MANUSCRIPTS

Submit only the original copy of the article, but retain a copy for use in correcting galley proofs. Carbon copies are not acceptable for editing and typesetting purposes.

REFERENCES

References should be placed at the end of the manuscript and should conform to the style of the Quarterly Cumulative Index Medicus. No more than twenty references should be included with a manuscript.

ILLUSTRATIONS

Only reproducible illustrations will be accepted. All photographs must be black and white glossy prints, clear cut and contrasting. The size of each photograph submitted should be no larger than 2 3/4" x 2". Instructions for combining photographs are acceptable. The Editorial Board reserves the right to limit the number and size of illustrations used with any manuscript submitted for publication. Any number of illustrations over three (3) is charged to the author.

REPRINTS

Reprint order blanks with a schedule of prices will be attached to galley proofs. Individual reprints of an article must be obtained direct from the author.

COPYRIGHT

Matter appearing in the ARCHIVES OF PHYSICAL MEDICINE AND REHABILITATION is covered by copyright. Permission for reproduction must be directed to the Editorial Board.

Detailed information covering instructions for the preparation of manuscripts for publication may be had by writing direct to the Editorial Office, 30 N. Michigan Ave., Chicago 2.

PHYSIATRIST — to assume medical direction of rehabilitation center for children and adults. Must be Boarded or Board eligible.

PHYSICAL THERAPIST — to direct physical therapy departments in children and adult centers. Must be fully accredited. Experience supervision students and staff; administration of Physical Therapy Department essential.

RESEARCH COORDINATOR — Training and experience in physical medicine and rehabilitation. Ph.D. or M.D.

For details write —

BOX 2611
ARCHIVES OF PHYSICAL MEDICINE
AND REHABILITATION



california calling

**GRADUATES OF APPROVED
SCHOOLS OF PHYSICAL THERAPY**

**Openings in State Hospitals • Veterans' Home
Crippled Children Services Program**

Starting salaries \$458 and up; liberal employee benefits; promotional opportunities. Streamlined civil service examinations twice a month in San Francisco and Los Angeles and on request in other states near candidate's residence.

Apply: STATE PERSONNEL BOARD, Dept. PT 70
801 Capitol Avenue
Sacramento 14, California

PHYSICAL THERAPISTS

Supervisory and staff openings. California licenses or eligible. New Rehabilitation Center. Write: Personnel Director, Cedars of Lebanon Hospital, 4833 Fountain Ave., Hollywood 29, Calif.

Registered or licensed physical therapist with special training and/or experience with cerebral palsied children to conduct established mobile unit service in Ohio. Occupational therapist also on service. Orthopedic medical direction. Station wagon and travel expenses provided. Forty-hour week; social security; three week vacation plus eight holidays, 10 days annual sick leave and five days annual educational leave with salary; annual increments. Salary up to \$6000 for a well qualified therapist. Apply Miss Grace Roberts, Cerebral Palsy Program Director, Ohio Elks Association, 580 E. Town St., Columbus, Ohio.

REGISTERED PHYSICAL THERAPISTS

To help staff of two year old, fully equipped department. Fully accredited hospital. Salary open. Generous fringe benefits. Address inquiries to Personnel Office, Evangelical Deaconess Hospital, 4229 Pearl Road, Cleveland 9, Ohio.

PHYSICAL THERAPISTS

Positions available for qualified physical therapists for active Rehabilitation Department in modern 500 bed, chronic disease hospital. Salary open. Four weeks vacation. Sick benefits, lunches. Apply Personnel, St. Barnabas Hospital, New York 57, N. Y.

Patronize

(Y)our

Advertisers

PRESTON

A Complete Line

FOR PHYSICAL MEDICINE AND REHABILITATION

Your one source for all your needs

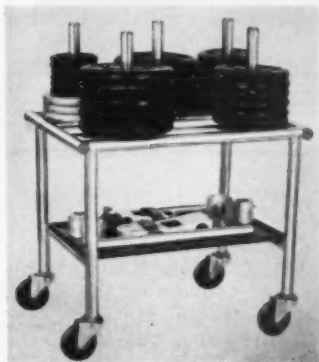


THE ALL-NEW PRESTON CATALOG NO. 1065

The Most Complete Catalog in the Field

Describes the leading and most complete line of Equipment for Rehabilitation Exercise, Hydrotherapy and Electrotherapy. Also: Diagnostic Apparatus, Traction Devices, Cerebral-Palsy Furniture, Wheelchairs, Walkers, Lifters, Crutches and Self-Help Devices. Includes over 2,500 selected items.

All orders sent to Preston Headquarters receive personalized prompt service—usually same-day shipment—from our well stocked warehouse. Whether you need a single article or a complete installation you can buy with confidence when you order from Preston. Catalog requests on your letterhead should be addressed to J. A. Preston Corporation, Department A.



THE PRESTON WEIGHT CADDY

Saves You Effort, Time and Space

No more storage problems, no more bending and carrying heavy weights by hand to the patient. The Weight Caddy allows the weights to be conveniently stored in any corner or closet, in a minimum of space. It can be wheeled quickly and without effort to treatment table. Requires only two square feet of floor space!

The Preston Weight Caddy is built to seat level, the most convenient height for the administration of a weight program. It is no effort at all for the therapist, sitting on a chair or stool, to apply weights to exercise boots.

Order by Catalog Number:

PC 2043—Preston Weight Caddy—on 3" ball-bearing casters with five weight holders and utility shelf, all-welded chrome plated construction, 20½" x 15" x 26" overall height.....\$54.50



THE STRYKER "CIRCOLECTRIC" HOSPITAL BED

Simplifies Patient Handling

Combines the features and advantages of an electric hospital bed, a standing bed, standing table and a turning frame. Problems such as lifting, turning and transfer of patients are eliminated.

The "CircOlectric" is especially helpful for all orthopedic cases, many neurological conditions: polio, muscular dystrophy, multiple sclerosis, cardiovascular diseases, and burns. Ideal for handling the aged and debilitated. Order by Catalog Number:

PC 4403—Stryker "CircOlectric" Universal Hospital Bed complete with mattresses, sheet set, siderails, footboard, 2 forehead bands, ½ H.P. silent motor, 84" long x 32½" wide x 78" high.....\$795.00

Optional Accessories are available at additional cost.

Send all orders to →

J. A. PRESTON CORP.

71 Fifth Avenue, New York 3, New York

HYDROCOLLATOR



Now moist heat can be applied conveniently, effectively and with a minimum investment in equipment. No dripping, no wringing, no repeated applications. Each application gives at least 30 minutes effective moist heat. The Steam Pack is merely heated in water, wrapped in a towel, and applied. Standard equipment in leading hospitals and clinics across the nation.

Folder and prices on request

Originated and Manufactured by

CHATTANOOGA PHARMACAL CO., INC.

CHATTANOOGA 5, TENNESSEE

MASTER UNITS: Five stainless steel models for various requirements in hospitals, clinics, physicians' offices, and patients' homes. Automatically maintain Steam Packs in water at proper temperature—always ready for use. No plumbing used.

READER SERVICE . . .

this section is established for the convenience of the reader-audience of the ARCHIVES OF PHYSICAL MEDICINE AND REHABILITATION. Each month, the reader service column will list the complete name and address of the author to be contacted for information and/or reprints of scientific studies herein published. The column will be up to date at press time, but the ARCHIVES cannot guarantee the accuracy of such listings 30 days after publication date. The following authors, in Contents order, are represented in the April, 1961 issue:

- ROBERT L. BENNETT, M.D., Executive Director, Georgia Warm Springs Foundation, Warm Springs, Georgia.
 STELLA Y. BOTELHO, M.D., G.S.M. Physiology, University of Pennsylvania, Philadelphia 4, Pennsylvania.
 HERBERT FISCHER, M.D., Rehabilitation Institute of Chicago, 401 E. Ohio Street, Chicago 1, Ill.
 DR. RONALD HARRIS, Devonshire Royal Hospital, Buxton, England.
 O. LEONARD HUDDLESTON, M.D., 1 Pico Boulevard, Santa Monica, California.
 OSVALDO E. MIGLIETTA, M.D., Department of Physical Medicine and Rehabilitation, New York Medical College, 1 E. 105th Street, New York 29, New York.
 CHARLES D. SHIELDS, M.D., Department of Physical Medicine and Rehabilitation, Georgetown University Hospital, 3900 Reservoir Road, N.W., Washington 7, D. C.
 DR. BRYNJULF STRANDBERG, Copenhagen County Hospital, Hellerup, Denmark.
 ODON F. VON WERSOWETZ, M.D., Medical Director, Texas Rehabilitation Center, Gonzales, Texas.
 JOHN S. YOUNG, M.D., 1599 Ingalls Street, Denver 15, Colorado.

Please do not direct requests for reprints to the Archives of Physical Medicine and Rehabilitation

NEW ELGIN EXERCISE CHAIR FOR PROGRESSIVE RESISTANCE EXERCISES

Here is an ENTIRELY NEW EXERCISE UNIT, clinically proven to be extremely beneficial during early treatment of the hemiplegic patient (reciprocal motion exercises for arms and legs). The NEW ELGIN EXERCISE CHAIR also provides excellent treatment for many other cases such as polio, arthritis, and the orthopedic patient.

May be used in the physical therapy department, hospital ward, or for home treatment.

Complete brochure illustrating the many exercises possible with this revolutionary new unit will be sent on request.

Elgin offers the most complete line of Progressive Resistance Exercise Equipment. Write today for free catalogues.



SOME FEATURES

- Sturdy . . . Safe Comfortable
- Requires Minimum Amount of Space
- Correct Mechanical Position While Exercising
- Excellent for Resistive, Assistive, or Reciprocal Motion Exercises

Elgin

EXERCISE APPLIANCE CO.
P. O. BOX 132 ELGIN, ILLINOIS

LaBERNE Utility Electric "WALK-OFF" TABLE

The LaBerne electrically operated Utility Model table is built of tubular steel and reinforced angle iron finished in Mellotone grey and mounted on 3" swivel casters with locks. Electrically operated by motor and gear box through a direct worm drive with reversing switch; safety switches for up and down positions.

Table is 78" long, 28" wide, and 32" high, adjustable to any position from horizontal to vertical with calibrated dial showing degree of tip from 0 to 90.

Fixtures included foam top covered with water proof leatherette; removable footboard, two 6" restrainer straps, and cervical hook.

Model 1060 E \$395.00

Hand Operated Model (photo below)

Same as above except operated by hand crank

Model 2101 H \$239.50

Also available are Standard Hospital Model, Clinic Model, Intermediate Model and Telescopic Model.

Prices FOB Columbia, S. C.

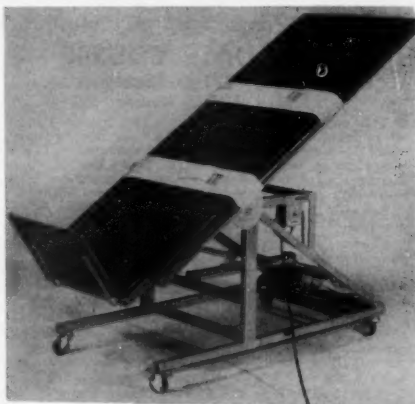
Write for catalog.

La Berne MANUFACTURING COMPANY

PO Box 5245 Columbia, S. C. Phone SU 7-6162



Originators of the "WALK-OFF" Physical Therapy Table



TREAT EDEMA, VASCULAR PROBLEMS with THE JOBST TREATMENT PROGRAM

Dramatic successes in reducing edema, in combatting vascular insufficiencies, and in improved stump therapy are being recorded with the Jobst Treatment Program.

First, the Jobst Intermittent Compression Unit is used. This reduces edema by gentle, pneumatic massage. The physician prescribes the desired compression and the time interval for pressure and rest.

When the edema has been reduced, custom-fitted Jobst Venous Pressure Gradient Supports are worn to maintain the benefits obtained. For most cases of vascular insufficiencies, Jobst Venous Pressure Gradient Supports alone are beneficial.



For details and clinical literature, write to

THE R. D. GRANT COMPANY

761 Hippodrome Building
Cleveland 15, Ohio
on Jobst Intermittent Compression Units

THE JOBST INSTITUTE

1805 Jefferson Ave., Toledo 2, Ohio
on Jobst Venous Pressure Gradient Supports



Georgia Warm Springs Foundation GRADUATE COURSE

Physical Therapy and Occupational Therapy
In the Care of Neuro-Muscular Disease

This course is open to graduates of approved schools of physical and occupational therapy. Such graduates must be members of the American Physical Therapy Association and/or registrants of the American Registry of Physical Therapists, or members of the American Occupational Therapy Association.

Entrance Dates: First Monday in January, April and October.

Course I — Emphasis on care of convalescent neuro-muscular disease with intensive training in functional anatomy, muscle testing, muscle reeducation and use of supportive and assistive apparatus. This course is complete in itself.

Course II — Three months duration with Course I prerequisite. Emphasis on care of severe chronic physical handicaps with intensive training in resumption of functional activity and use of adaptive apparatus.

In-Service Training Program — Fifteen months duration at salary of \$225 per month plus full maintenance, increasing to \$250 per month at the completion of nine months. This program includes training in Courses I and II.

Tuition: None. Maintenance is \$100 per month (except those on In-Service Training Program). For scholarship to cover transportation and maintenance for Courses I and II, contact The National Foundation, 800 Second Avenue, New York 17, New York. (Scholarships require two years of experience.)

For further information contact:

ROBERT L. BENNETT, M.D.

Executive Director

Georgia Warm Springs Foundation
WARM SPRINGS, GEORGIA

WATERS MS-12 **MUSCLE STIMULATOR**



- DESIGNED FOR HOME USE
- LOW IN COST
- SIMPLE TO OPERATE
- RELIABLE GALVANIC OPERATION

WATERS CORP.

P. O. Box 288 • Rochester, Minnesota

FROM: TECA CORPORATION

Devoted to development of electronic instrumentation for neuromuscular studies.

A NEW PORTABLE ELECTROMYOGRAPH, incorporating all facilities for nerve conduction time tests and Polaroid® photo-recording. The new Model B has been developed to supplement the 1 and 2 channel console Models TE1.2-7 and TE2-7. A matching console cabinet for the Model B can include an extended range magnetic tape recorder for operation with the EMG. The new portable, a compact one channel unit, will find application where the expandability, 2 channel facility, and flexibility of the TE console series are not required or where mobility is a prime requirement.

NEW NERVE CONDUCTION TIME FACILITIES in the Model B include unique time marker pulses superimposed electrically on the recording beam, permitting accurate direct measurement of conduction time, without error due to parallax or sweep and cathode ray tube distortions.

The stimulator, which generates rectangular pulses with 5 adjustable calibrated duration settings, provides high maximum output voltage (250V). The unique mounting of the intensity control on the percutaneous stimulator electrode handle now makes testing easier. Frequency of stimulus is adjustable from 1 per second to well into the tetanus range. An accessory conduction time indicator will permit direct reading of conduction time (or other physiological time intervals) from a digital indicator calibrated in milliseconds.

OTHER USEFUL FEATURES include a rugged Polaroid® camera mount that permits the camera to be instantly swung into position ready for use.

All electronic camera synchronization is fast, foolproof and easy to use for EMG or conduction time recording. The seven inch diameter flat face cathode ray tube is photographed thru an illuminated screen with engraved scale.

Advanced circuitry includes regulated power supplies for stability and accurate calibration, at low and high power line voltages. Radio frequency rejection circuits and 60 cycle filters are provided.

OTHER NEW DEVELOPMENTS

An electromagnetic percussion hammer delivers uniform repeatable impacts to the body. The impact is synchronized with the sweep of the EMG so that muscle response, either electrical (EMG) or mechanical (motion velocity or acceleration) may be displayed as a stationary pattern on the screen, permitting accurate time measurements to be made, visually and by magnetic tape or photo-recording.

Continuous record high frequency multichannel photo-recorders for automatic operation in conjunction with the EMG. Permanent graphic records are provided with wide range of time scales and with integral time markers.

Electrical integrators for muscle potentials, with both averaging (adjustable decay) and absolute integral modes of operation.

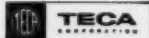
Special stimulators for operation synchronized with the EMG, supplying electrically isolated pulse pairs with adjustable delay between each of the two independent outputs. Isolators can be supplied to users of Teca Chronaximeters to permit EMG recording of evoked potentials.

Also for electrodiagnosis, the Teca Model CH-3 a true stabilized current variable pulse generator and chronaximeter. Generates accurate rectangular pulses of current entirely independent of variations in patient or electrode impedance over a wide range. Incorporates many innovations for simplified accurate strength duration curve and chronaxie procedure. Write for Bulletin.



New Portable Model B with camera in retracted position.

80 MAIN STREET, WHITE PLAINS, NEW YORK



ASK (or tell) THE MAN WHO OWNS ONE



We just have completed a mailing program that I think once again points up the efficacy of Physical Medicine to the general medical profession. (Before I go any further, my secretary has already questioned the use of the word "efficacy." I showed her in the Webster that it merely meant "... power to produce effects," but she was still sullen. Anyway, who's my secretary to be always pushing me around? I'll use it if I want to . . . efficacy, *efficacy*, EFFICACY xx!!)

If you'll wipe away those tears, Mrs. Moore, let's continue.

The mailing program I referred to above was a simple two-part business reply card; one half showed some of the current applications of Ultrasonics you folks are using and the other half a typical "business-reply" card by which the person could get some of the published reports covering Ultrasonics in the general medical office. We mailed these cards to the General Practice MD's in the country.

Well, if anyone ever had any doubts about how MD's felt about Physical Medicine, I think the return of cards to us can certainly dispel the questions. We were literally swamped by cards from doctors in every state . . . and our mailing department burned a little midnight oil to make sure

that every one of the requests for information were filled. Clay Williams, who's in charge of our Physical Medicine Division, still flinches when a mailman walks by.

What brought this to mind this morning was a report I received from Clay covering the fact that over 40% of doctors in general practice are now using Ultrasonics, according to a recent Medical Journal national survey. It just seems to me that folks like you and us, who have spent a good many years in the Physical Medicine field, may have an obligation to our profession to give a hand to these men whenever we can. Obviously we can't go around and give treatments, but we *can* work on whatever level is available so that general practitioners get as much information as possible on the subject.


Perhaps there is a GP in your immediate area who feels that he is not getting entirely favorable results with his Ultrasonic Therapy. It might be a worthwhile effort to spend just a few minutes in teaching him what the method is all about, and how to do it. Such time spent might not mean any additional dollars in your pocket, but I believe it serves a greater purpose . . . that of providing an additional support in the building up of the Physical Medicine profession to the level to which it can and should aspire.

Cordially,

Cecil Birtcher, President
The Birtcher Corporation

THE BIRTCHER CORPORATION

4371 Valley Boulevard, Los Angeles 32, California

One Quarter Century
Of Honest Value
Sincerely Presented  B



Rx SPENCER

Post surgical embolisms following a hysterectomy for carcinoma of the uterus resulted in general paralysis and acute paresthesia of this patient's right side.

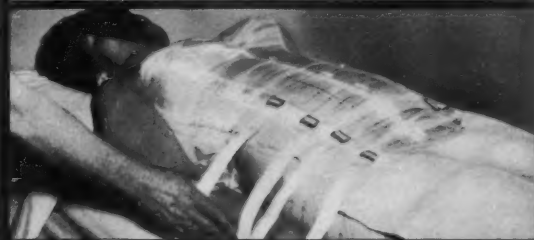
Because of extensive muscular deterioration, she was confined to a wheelchair and only recently (following a rehabilitation program) has become ambulatory with the aid of crutches.

As an adjunct to rehabilitation and to improve her general health, elevation of patient's severely ptosed abdomen and proper bracing of her partially paralyzed trunk, without constriction of thighs, was necessary.

A Spencer was designed to meet the varied support needs of this patient.

why Spencer?

- A Spencer Support meets *all* of the patient's support requirements because it incorporates the features required in the basic style support indicated by the patient's figure needs.
- Individually designed for the patient who is to wear it, a Spencer Support is made to give the exact degree and type of support required, from extreme flexibility to the immobilization achieved in a brace.
- Spencer first realigns the body (using the patented control support shown in the photograph below) . . . then designs a support to hold it that way.



Spencer's Individual Designing Service is available to you through Corsetieres specially trained to help you help your patients.

SPENCER

*individually designed supports
for women, men and children*

SPENCER, INCORPORATED

35 Ellsworth Ave., New Haven 7, Conn.

Canada: Spencer, Ltd., Rock Island, Quebec

England: Spencer, Ltd., Banbury, Oxon

- ☐ Send me the name of the nearest Spencer Corsetiere.
- ☐ I would like an office demonstration of Spencer benefits and features.

Name

Address

4-61



You buy more than a good return with Savings Bonds. You help keep our country strong for today's generation and the ones to come. That's one of the reasons so many Americans buy and hold U. S. Savings Bonds.

How to grow your own money

A U.S. Savings Bond is better than money. It grows. Plant \$18.75 in a Savings Bond and in 7¾ years it'll be \$25—a full one-third bigger. The U.S. Government guarantees this growth. And if you can't afford to lay out the full purchase price, you can buy Savings Bonds a bit at a time through your Payroll Department where you work. It's an automatic installment plan that costs you no interest or carrying charges.

Advantages to think about

You can save automatically on the Payroll Savings Plan • You now earn 3¾% to maturity • You invest without risk • Your Bonds are replaced free if lost or stolen • You can get your money with interest anytime you want it • You buy shares in a stronger America.

You save more than money with U.S. Savings Bonds



This advertising is donated by The Advertising Council and this magazine.



R_x For the practice of modern medicine

THE ALL NEW ELECTROMYOGRAPH By MEDITRON

AUTOMATIC
PHOTOGRAPHY

HIGH IMPEDANCE
PREAMPLIFIER



CONTROLLED SWEEP
7" OSCILLOSCOPE

NERVE CONDUCTION
VELOCITY STUDIES



IMMEDIATE DELIVERY — FULLY GUARANTEED — WRITE OR TELEPHONE COLLECT TODAY

THE MEDITRON COMPANY

708 S. FAIROAKS, PASADENA, CALIFORNIA



CENTER STREET, PEMBROKE, MASS.

A MUST IN DERMATOLOGY FISCHERQUARTZ



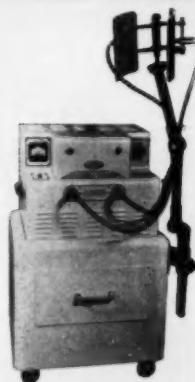
Model No. 88

R. A. FISCHER & CO.

Eastern Office and Warehouse: HABER INC., 2043 CHESTNUT ST., PHILADELPHIA 3, PA.

FISCHERTHERM • FISCHERSINE • FISCHERQUARTZ • FISCHER ULTRA SOUND

A MUST IN DIATHERMY THE MASTER FISCHERTHERM



Model No. 1200 — A powerful, dependable machine for continuous performance, night & day.

FISCHER DRY PADS FOR LOW VOLT
NO MOISTURE
SEND FOR DETAILS

517-525 COMMERCIAL STREET
GLENDALE 3, CALIFORNIA

Enjoy a wealth of
INFORMATION
and new ideas . . . all yours
WITHOUT COST!

Write for
CATALOG PM
picturing and describing
our new, improved
SURGICAL and ORTHOPEDIC
APPLIANCES
for early training and
REHABILITATION



Note Cosmevo's change of name and address

COSMO ORTHOPEDIC
MFG. CO.
236 River St., Hackensack, N. J.

NEW HAND CONTROL FOR CARS
ECONOMY MODEL *Handi-Drive*

APPROVED FOR SAFETY
By State Highway Commissions

There is a new, improved hand control for cars, designed by a Swedish craftsman. It is made of solid aluminum, is durable, neat in appearance and light in weight. There is nothing to clutter up the dash. Others can drive with equal ease.

with
HANDI-DRIVE
You don't need to
use your feet to drive

GUARANTEED
One to 30 days - if not satisfied -
your money back - no questions asked

Manufactured By:
THE LEVERING HAND DRIVE CO.
Box 873 Evan, North Dakota

PUSH AHEAD FOR GAS
PULL BACK FOR BRAKE

PRICE
\$97.50
CASH PAID

Here is a new, improved hand control for cars, designed by a Swedish craftsman — bringing you streamlined equipment both attractive and efficient. Made of solid aluminum, it is durable, neat in appearance and light in weight. There is nothing to clutter up the dash. Others can drive with equal ease.

massages
pain away
in **musculoskeletal**
involvements

GER-O-FOAM

(aerosol foam)

relieves pain, spasm;
improves function
increases tolerance
to exercise



GER-O-FOAM's exclusive formula provides for the first time deeply absorbed analgesic-anesthetic agents in aerosol form—to permeate and anesthetize sensory nerve endings.

Relief in minutes, lasting for hours in . . . rheumatoid arthritis, osteoarthritis, muscle sprain, fibromyositis, low back pain . . . even in chronic intractable cases.

GER-O-FOAM combines: Methyl salicylate 30%, benzocaine 3%, in a neutralized emulsion base, permitting fast penetration through the stratum corneum.

Sample

and reprint from

GERIATRIC PHARMACEUTICAL CORP.
Bellerose, New York

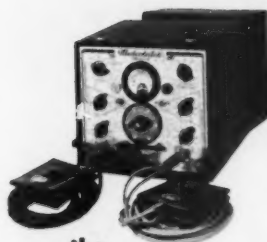
Pioneers in Geriatric Research

I. Gordon, E. E. and Haas, A.:
Industrial Medicine & Surgery
28:217, 1959.



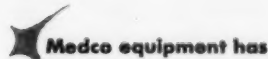
THREE QUALITY MEDCO PRODUCTS

to save you **TIME** and **MONEY** . . . and
EASE YOUR PERSONAL WORK LOAD

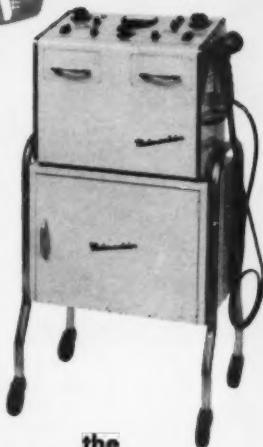


the
Medcolator
MODEL G
with **GALVANIC**

Has alternating, interrupted, modulated current of high or low frequency, superimposed on a rectified current. It incorporates three settings, Pulse, Surge and Tetanizing. The galvanic current and the Medcolator current may be used individually or simultaneously. Two other models also available.



Medco equipment has
wide medical acceptance by individual physicians,
clinics, hospitals and industrial plants.



the
Medco-sonlator

Provides complete facilities for combination of Electrical Muscle stimulation and Ultrasound in one convenient cabinet. The use of Electrical Muscle Stimulation and Ultrasound, simultaneously applied, through the same sound head applicator, offers a diagnostic as well as combination therapy not possible with either therapy individually.



the
Medco
KOL-THERM

Provides an entirely **new** approach to one of medicine's oldest therapies, producing instantaneous moist or dry cold or moist or dry heat. The temperature range extends from approximately 30° to 130° with contrast therapy available and with completely automatic operation.

Now, the new **KOL-THERM**, for the instantaneous application of heat or cold, is also receiving an enthusiastic welcome. Medco trained personnel works with each physician and his assistants teaching them the operation of the equipment until it becomes second nature. The operation of the equipment by an assistant relieves the physician of a heavy portion of his daily work load.

A convenient coupon, at the right, may be marked and mailed for complete information or an office demonstration of any Medco equipment.



"Serving the Profession Since 1932"

MEDCO ELECTRONICS COMPANY, INC.

Division/Medco Products Co., Inc.

3401 E. Admiral Pl.
Dept. APMR 21
P. O. Box 3338
Tulsa, Oklahoma

- ☐ I would like an office demonstration of your.....
☐ Please send me your illustrated literature on the.....

NAME.....
ADDRESS.....
CITY.....STATE.....



...THEY CAN WALK AGAIN

Torpedoed on the Murmansk run — nearly frozen to death in an open boat — both legs lost below the knee — ex-Merchant Marines Michael McCormick and William Morris walked unaided in three weeks. They could look forward with certainty to leading a normal life again. To these men, as to thousands of other Hanger wearers, the phrase "Hanger is a symbol of help and hope" is a concrete truth proven by every day of their future lives.

Hanger
PROSTHESES

AVAILABLE AT AUTHORIZED FACILITIES IN THE FOLLOWING CITIES:

Eastern Region:

Baltimore 1, Md.
Boston 15, Mass.
Charleston 1, W. Va.
New York 11, N. Y.
Philadelphia 7, Pa.
Raleigh, N. C.
Richmond 19, Va.
Roanoke 12, Va.
Washington 13, D. C.

Central Region:

Columbus 8, Ohio
Pittsburgh 1, Pa.

Midwestern Region:

Chicago 5, Ill.
Cincinnati 29, Ohio
Dallas 1, Texas
Evansville, Ind.
Fort Wayne, Ind.
Indianapolis 2, Ind.
Oklahoma City 3, Okla.
Peoria 4, Ill.
St. Louis 66, Mo.

Southeastern Region:

Atlanta 9, Ga.
Birmingham 1, Ala.
Columbia 1, S. C.
Jacksonville, Fla.
Lafayette, La.
Miami 37, Fla.
Mobile, Ala.
Montgomery, Ala.
Nashville 5, Tenn.
New Orleans 19, La.
Orlando, Fla.
Savannah, Ga.
St. Petersburg, Fla.
Tampa 2, Fla.
West Palm Beach, Fla.

NEW PRODUCTS FROM

Burdick

RESEARCH

ELECTRO-
CARDIOGRAPH



ULTRASOUND



DIATHERMY



To make sure that Burdick electromedical equipment is always among the finest available, our research and engineering departments constantly strive to improve existing units and to develop new products.

ELECTROCARDIOGRAPH — The new dual-speed EK-III has been given enthusiastic acclaim by doctors everywhere, providing either 25 mm. or 50-mm.-per-second paper speed — for more accurate diagnosis under the most difficult situations.

ULTRASOUND — The new UT-400 provides the utmost in ultrasonic versatility—either continuous or pulsed ultrasound is produced.

DIATHERMY — The Burdick MF-49 unit offers a compact yet effective means for every short wave application. For microwave diathermy it's Burdick's popular MW-1 Microtherm.*

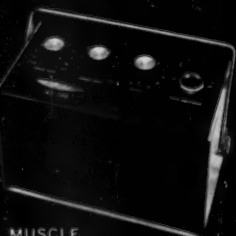
MUSCLE STIMULATOR — The new MS-300 is ideal for electrical stimulation of innervated muscle tissue. It provides effective therapy for the individual condition treated, without patient discomfort.

INFRARED — The Zolite series has become a standard of quality and performance for the hospital, physician's office and home use on prescription.

CARDIAC MONITOR — The new TC-20 Telecor monitors heart beat during surgery, either electrically or mechanically. An invaluable instrument with many applications.

THE BURDICK CORPORATION
MILTON, WISCONSIN

Branch Offices: New York • Chicago • Atlanta
• Los Angeles • Dealers in all principal cities



MUSCLE
STIMULATOR



INFRARED

CARDIAC MONITOR

MISS PHOEBE

NO. 40 IN A SERIES



"Can it truly be that the wondrous chair of Everest & Jennings weighs less than the magic rope of hemp?"



Everest & Jennings chairs *are* lightweight—yet no wheel chair on the market is stronger or has better balance. Everest & Jennings' modern good looks and effortless handling overcome "wheel chair shyness" and invite activity. For patients young or old, you can recommend an Everest & Jennings with confidence.



Rust-proof SHOWER CHAIR
doubles as over-toilet commode;
seat and back withstand
200-degree boiling water.

There's a helpful authorized dealer near you

EVEREST & JENNINGS, INC., LOS ANGELES 25

Recognition and Care of Early Scoliosis

Robert L. Bennett, M.D.
Warm Springs, Ga.

● This paper discusses two common forms of scoliosis; paralytic and idiopathic. All physicians who care for scoliosis must accept that structural deformity of the vertebrae and the rib cage cannot be altered to any significant degree by conservative, or even surgical procedures. For this reason, in individuals who have not reached skeletal maturity, all lateral and rotary deviations of the spine, regardless of how mild they appear to be, deserve deliberate and persistent treatment. If these beginning curvatures are recognized, most of them can be adequately controlled, and structural deformity kept at a minimum. The prevention of severe scoliosis is possible only if the physician is aware of the causative and accelerating factors that may be responsible for the development of a severe scoliosis. A curve must be considered structural and, therefore, dangerous when the spine shows asymmetrical mobility. Both hyper- and hypomobility are equally dangerous when asymmetrical. Combination types exist and have their characteristic patterns of progression. Obviously, all types of scoliosis cannot be prevented and treated in the same way and, while care must be strictly individualized, there are certain basic requirements common to all. All abnormal and asymmetrical stress against the proper alignment of the spine must be controlled insofar as possible. This will require such simple measures as weight and activity control, but might also require specific surgical measures such as release of iliotibial fascial contractures, reinforcement of the abdominal wall with fascial strips, and even early local fusion of the vertebrae. In general, it can be said that gymnastics have no place in the care of scoliosis.

It will be the attempt of this presentation, first, to emphasize that early recognition of persistent lateral and radial deviations of the spine is necessary if we wish to prevent the development of dangerous and unsightly structural changes in pelvis, vertebrae, and thoracic cage; next, to review a simple classification of early scoliosis, and the possible causative and accelerating factors responsible for the four types in this classification; and, finally, to discuss the basic principles of prevention and correction of early scoliosis.

If a scoliosis is to be prevented, the physician must not only constantly anticipate, search for, and treat early lateral deviations of the spine but also be capable of recognizing all factors that could possibly bring about persistent faulty alignment of the spine. The physician must be aware that an idiopathic scoliosis can develop in otherwise completely normal boys, as well as girls, during the entire span of their growing period. He must expect the development of a scoliosis in all of his patients with neuromuscular and musculoskeletal diseases. Only in this way can very early deviations of the spine be found and

controlled at a time when control is possible. Each severe structural scoliosis begins as a mild and usually intermittent deviation from normal alignment. As the deviation, regardless of cause, becomes more persistent, secondary changes take place in the tissues supporting the spine, and general bodily patterns of activity are adapted to this persistent faulty position of the spine. Eventually, irreversible structural distortion occurs.

Clinical examination of the spine is not difficult if done in an orderly manner. It should be initiated by determining the persistent relationship of each iliac crest to the thoracic cage. This must be observed in the lying, sitting, and standing positions. At the same time, rotation of the pelvis — backward or forward — as related to the thoracic cage can be noted.

Lateral mobility of the lumbosacral region must then be determined. Normal mobility of the fourth and fifth lumbar vertebrae on the first sacral vertebra rarely exceeds 15 degrees to the right and to the left. Any evidence of asymmetrical contracture or hypermobility in this region is highly significant. It should be emphasized repeatedly that hypermobility of the spine, if asymmetrical, is just as important and dangerous as is asymmetrical contracture.

The alignment and mobility of the spine above the lumbosacral junction is then tested. Above the lumbosacral region it is more important to evaluate diagonal anterolateral movements than purely lateral ones. In this way, the earliest evidences of contracture or hypermobility in those structures attached to the posterior processes of the vertebrae is discovered. It would appear that asymmetrical changes in these structures are mainly responsible for the dangerous rotation of the vertebrae as lateral

Executive Director, Georgia Warm Springs Foundation, Warm Springs; Professor of Physical Medicine and Rehabilitation, Emory University Medical School, Emory University, Ga.

Read at the 3rd International Congress of Physical Medicine, Session on Scoliosis, Washington, D. C., August 23, 1960.

deviation takes place. It should not be necessary to emphasize that rotation of the bodies of the vertebrae towards the side of the convexity of the curve causes prominences which should not be mistaken for muscle hypertrophy. It should also be pointed out that marking the tips of the spinous processes of the vertebrae is a very inadequate way of determining the degree of scoliosis. X-ray examination of the spine is essential to discovery of very early deviations and in determining the reaction of the spine to weight bearing and other stress conditions. While x-rays of the spine may be taken in the lying, sitting, standing positions, the sitting position appears to be by far the most practical and informative. The sitting position usually can be assumed for a temporary period by all patients, regardless of the extent of paralysis. In this position, the alinement of the spine can be observed during its attempt to support the weight of the head, upper extremities, and chest.

Whether or not the spine is found to be malaligned, it is still necessary to search for all factors that might be capable of causing persistent lateral or rotary deviations of the pelvis, vertebrae, or thorax, and thus be capable of causing an existing curve, or capable of bringing about a persistent deviation in the future. While primary asymmetric changes within the body of the vertebrae and its posterior processes, or in the tissues immediately adjacent to the vertebrae, are of greatest significance in causing persistent deviation, any faulty positioning during rest or activity of bodily segments, even though quite remote from the spine itself, may cause deviation of the spine and eventual secondary changes in the vertebrae and the tissues immediately adjacent to it. These causes will be more specifically discussed under the classification of early scoliosis.

Patterns of Early Scoliosis, Their Possible Causes and Control

Paralytic disease, such as poliomyelitis, capable of involving the skeletal musculature of bodily segments in multiple localized or diffuse patterns, offers an ideal opportunity to observe the effect

of weakness or imbalance on the alinement of the spine. We seldom have a similar opportunity to observe the early changes in an idiopathic scoliosis. While all spinal curvatures of any significance eventually cause some degree of imbalance of muscular strength and use, there is no reason to believe that all so-called idiopathic curves are paralytic in origin. Muscular weakness superimposed on mild idiopathic structural faults is commonly seen. When this occurs it gives us an accelerated picture of the patterns of spinal distortion similar to those found in the idiopathic scoliosis.

It takes little experience with the problem of scoliosis to realize that a curvature of the spine may occur at any level for reasons that may or may not be obvious, and may progress, or occasionally regress, rapidly or slowly to an ultimate degree, apparently uninfluenced by either careful treatment or utter neglect. Fortunately, most idiopathic and paralytic curves begin as one of four easily recognized patterns of deviation from normal alinement and if discovered early can be controlled by specific care. In this discussion, these early deviations will be designated as Types I through IV. The order of listing does not refer to frequency or to significance, but rather to an arbitrary listing of the curve patterns beginning in the lumbosacral region and extending upward to the high dorsal. When the scoliosis is visualized early in its development, it usually can be assigned to one type. Combined types are common and expected in the later stages of development. An understanding of the pathogenesis and significance of the individual type enables us to analyze and treat the various combinations that may occur.

Type I

There are three prerequisite findings essential to the identification of this type:

1. The pelvis must be level, or very nearly so, when the patient is sitting or standing.
2. The lateral lumbosacral angle is more acute on one side than the other. This means that there is a lateral tilting,



Fig. 1 — Early Type I deviation.

or shifting of the fourth and/or fifth lumbar vertebrae on the first sacral vertebra. This is usually accompanied by limited mobility on the side of the acute angle and hypermobility on the opposite side.

3. A compensatory curve in the mid- or high lumbar region.

There appear to be at least five important causes of Type I. Any one cause may be found alone, or in combination with any of the others. Any one tends to cause the development of the others.

1. Pre-existing developmental faults in the lumbosacral region.

2. Weakness or imbalance of intrinsic spinal muscles in this region.

3. Weakness or imbalance of quadratus lumborum or ilio psoas.

4. Asymmetrical contracture of the iliotibial band. This contracture may be to the right or left of the acute angle.

5. Asymmetrical loss of bulk at the hip and thigh, or extremity length, particularly when on the side of the acute angle.

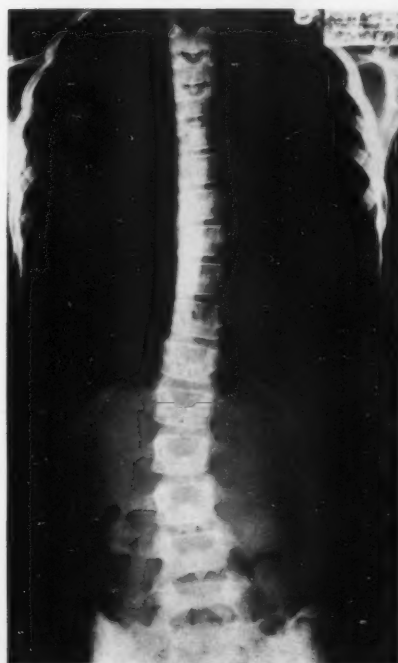


Fig. 2 — Lumbar progression pattern in Type 1 curve.

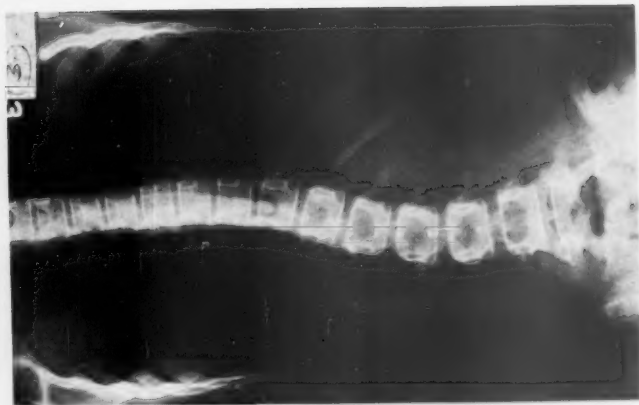


Fig. 3 — Dorsal progression pattern in Type 1 curve.

This type is important because faulty lateral takeoff at the base of the spine puts a strain on the entire spinal column and tends to exaggerate any weakness and imbalance that might exist above.

If the faulty lateral takeoff in the lumbosacral region is not corrected, one of two patterns of progression may occur.

(1) The faulty lateral takeoff will increase and the lumbar curve, secondary to this deviation, will progress rapidly. The dorsal spine will remain in good alignment except for its lower portion which will become the base of the lumbar curve. The rib cage will not be significantly distorted. This pattern is commonly seen in both paralytic and idiopathic scoliosis. (2) A compensatory curve will develop in the dorsal region to balance the lumbar curve. This dorsal curve may progress out of control, cause severe distortion of the thorax, and become the major problem. As the dorsal curve progresses, the lumbar curve — and even the lumbosacral angle — may be markedly corrected. This pattern also is frequently seen in both paralytic and idiopathic scoliosis.

Specific treatment of Type I should emphasize the following six points:

1. An immediate attempt should be made to mobilize the acute lumbosacral angle. This may be done by specific passive or active stretching. X-ray visualization of this region is necessary to determine the extent of the deviation and to determine the response to treatment. X-ray visualization also will determine if a structural fault exists that might limit the possibility of realignment of the spine, regardless of treatment.

2. If asymmetrical contracture of the iliotibial fascia exists, every attempt must be made to mobilize this band, either by conservative stretching or fasciotomy. Conservative stretching should be attempted first, but if release of contracture is not obtained in a reasonably short period of time, surgery must be carried out. It is important to realize that asymmetrical tightness of the iliotibial fascia is equally dangerous on either side. If it exists on the side of the deviation, it will tend to depress the pelvis, increasing the lumbar curve. If it is present on

the side opposite to the lateral deviation, it will tend to increase the hypermobility of the lumbosacral region on the opposite side.

3. A gluteal pad under the side of the acute lumbosacral angle should never be utilized without very serious consideration and x-ray study. It is always a temptation to realign the lumbar curve, secondary to the lumbosacral deviation, by lifting the pelvis on the side of the convexity of this lumbar curve. This usually results in an increase of the basic lumbosacral fault.

4. This type of early scoliosis cannot be controlled or even significantly altered by any type of back brace or corset. There is no way through trunk support to obtain corrective pressure low enough in the lumbar region to affect the lumbosacral angle. Spinal traction is of some limited value and should be utilized.

5. Any activity that causes downward pressure against the lumbosacral region should be sharply limited. This means that there should be marked restriction of both sitting and standing activities. The patient should be kept in a reclining or semi-sitting position as much as practical and consistent with the care of other skeletal problems.

6. The greatest danger of this type is the later development of a compensatory dorsal curve to the side opposite the lumbosacral deviation. This must be carefully watched for and kept under control even at the expense of permitting an increase in the lumbar curve.

Type II

This type is characterized by:

1. A pelvic obliquity (tilted pelvis) in the sitting or standing positions.
2. A normal lumbosacral takeoff.
3. A mid-lumbar curve with convexity on the side of the lower iliac crest.

This type must be separated into its two forms: the static and the dynamic.

Static Type II: This is a very common form of pelvic obliquity; it is simple to correct, and important only as it influences other types of deviations. It is most commonly caused by:

1. Asymmetrical loss of bulk in the hip and thigh, causing the pelvis to tilt when the patient is sitting.

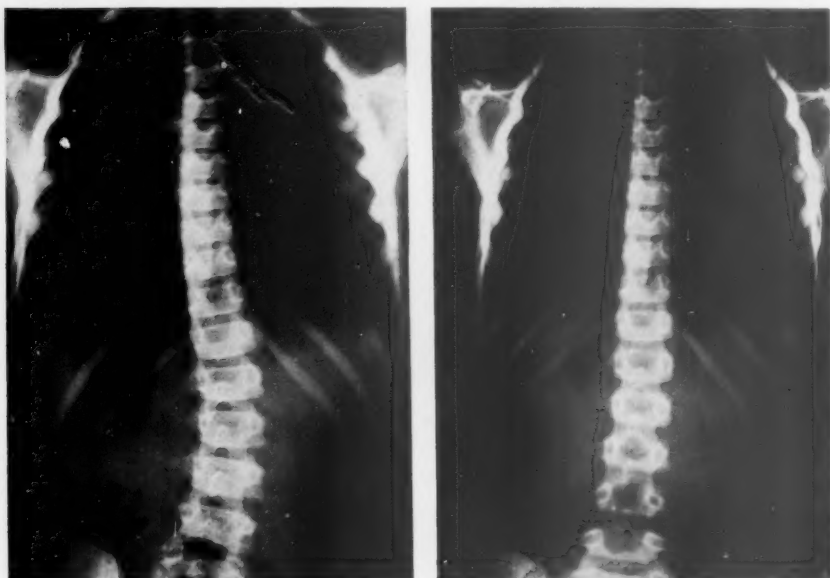


Fig. 4 — Static (Functional) Type II showing correction when pelvis is leveled by gluteal pad.



Fig. 5 — Dynamic Type II and usual pattern of progression.

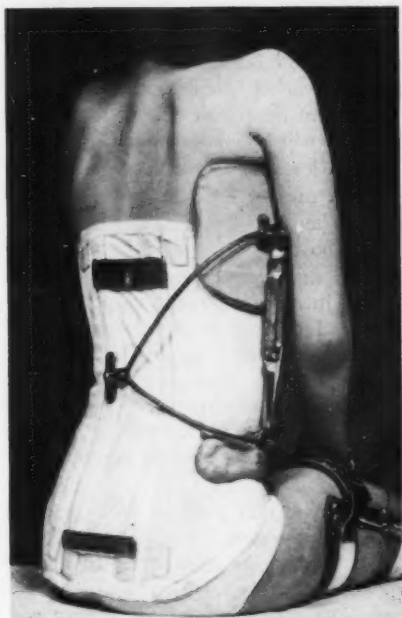


Fig. 6 — "Three-point pressure" corset used to correct dynamic Type II curve.

2. Discrepancy in lower extremity length, causing the pelvis to tilt when the patient is standing.

3. Faulty fitting of lower extremity apparatus may cause the patient to sit or stand with a tilted pelvis.

Tilting of the pelvis for the above reasons causes a simple curve that may be easily corrected by whatever means is necessary to bring about a level pelvis.

Dynamic Type II: This type is commonly referred to as a "dynamic pelvic obliquity." It is brought about by severe imbalance of strength of trunk musculature of hemiplegic pattern. The pelvis is kept in a persistently tilted position by the overuse, or the unopposed use of lateral trunk musculature lifting the pelvis upward, and pulling the thorax downward on the side of the stronger musculature. This imbalance is almost invariably coupled with iliotibial fascial contracture on the side of the weakened musculature. This contracture causes further tilting of the pelvis. This form is commonly complicated by subluxation, or actual dislocation of the hip on the side of the stronger trunk musculature.

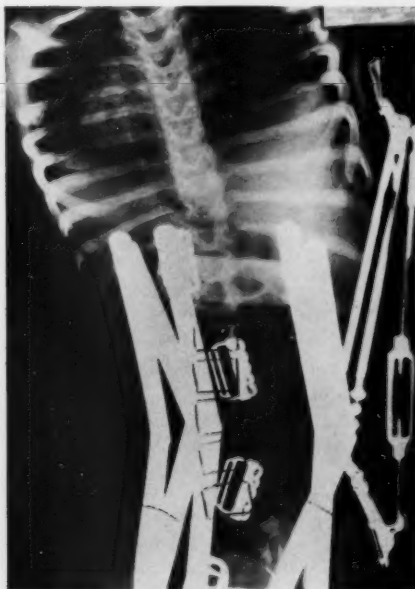


Fig. 7 — Correction of dynamic Type II following plaster wedging, fascial strip reinforcement of the abdominal wall, and support with three-point pressure corset.

At best, the treatment of this dynamic pattern is extremely difficult. The ilio-tibial fascia should be mobilized on the side of the trunk weakness and should be left slightly snug on the opposite side. A so-called "three-point pressure corset" was designed specifically for this type of scoliosis. This corset has an attachment which provides adjustable pressure pads downward against the iliac crest and upward against the posterolateral inferior costal margin on the side of the lumbar concavity. The corset provides the necessary pressure against the convexity.

Manual stretching of the tissues on the side of the lumbar concavity must be instituted early and carried out persistently and skillfully. Progressive wedging of this region by means of a plaster cast around the trunk is of great value and should be utilized early and repeated frequently. It is in this type of scoliosis that reinforcement of the weak side of the abdominal wall with fascial strips should be considered early.

Type III

This is a type common to both idiopathic and paralytic scoliosis, and is characterized by a so-called "long

C-curve" or "weak trunk curve." Maximum convexity is usually in the low dorsal region, but may occur at any level between the eighth dorsal vertebra and the second lumbar vertebra. In its pure form, it will be characterized by:

1. A level, or nearly level pelvis.
2. A normal lumbosacral takeoff.
3. The long "C-curve" mentioned above.

This deviation occurs when there is loss of intrinsic muscular support of the vertebral column in the lower half of the thoracic and upper half of the lumbar regions. The pattern of collapse is modified by asymmetrical strength and use of the extrinsic musculature of the trunk and adjacent pelvic and shoulder girdles.

This type may progress by rapid and severe progression of the basic dorso-lumbar curve in a manner frequently spoken of as "spinal collapse." Compensatory curves may occur above and below the primary dorsolumbar region and either one may progress out of control. Rarely can levels of convexity be directly related to specific muscular weakness. Hemiplegic involvement, particularly of lateral abdominals and

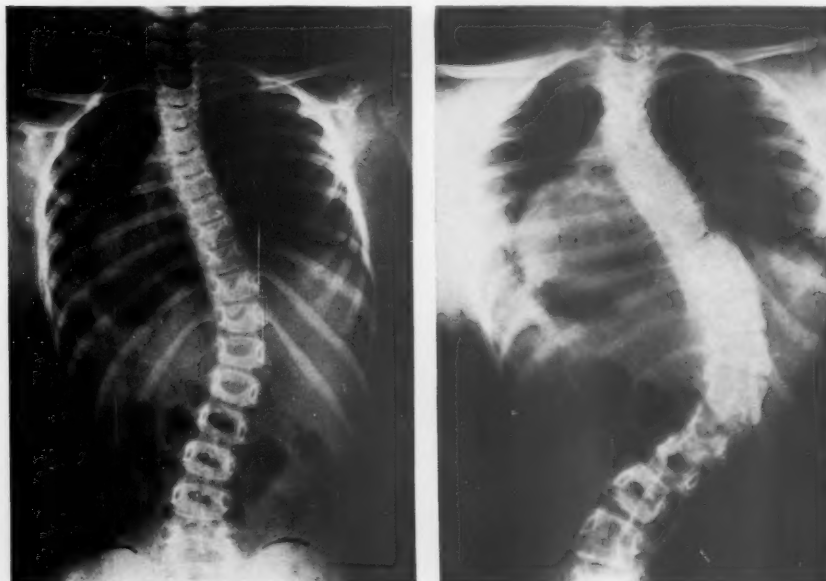


Fig. 8 — Type III and usual pattern of progression.

latissimus dorsi, may cause deviation to either side of the weakness. If the patient is kept in the reclining position, the curve is most likely to be convex on the side of the weakness. If he is permitted early sitting and standing, the curve is most likely to be convex on the side of the strength. When there is diagonal weakness in the lateral abdominals (weakness in one external oblique and the opposite internal oblique), the thorax is shifted toward the side of the weak external oblique.

In this type, the attempt is made to obtain and maintain symmetrical mobility of the spine. This does not mean that hypermobility, or even normal mobility is desirable in each case. If muscle weakness or imbalance exists, symmetrical tightness may be necessary to maintain alignment.

Many different types of spinal support have been designed for this type of curve. As will be mentioned below, we believe that a basic Hoke corset may be used with sufficient reinforcement to give the spine adequate support. This type of corset has the advantage that it may be shifted at its top to the right, or left, to

combat the tendency of the dorsolumbar curve.

Type IV

This type is identified by its angular curve at the level of the fourth, fifth or sixth dorsal vertebrae. In its pure form, it has the following characteristics:

1. A level, or nearly level pelvis.
2. A normal lumbosacral takeoff.
3. A long, mild dorsolumbar curve.
4. An angular curve, usually at the level of the fifth dorsal vertebra.
5. Very early flattening of the rib cage immediately opposite and below the convexity of the high dorsal curve.

The causes of this scoliosis are not clear. It would appear that weakness or imbalance of the intrinsic spinal musculature in the upper dorsal region must be at fault, but this weakness is extremely difficult to demonstrate. In many patients, there is very obvious loss of muscles of inspiration, particularly intercostal muscles on the side of the convexity. Asymmetrical weakness about the shoulder girdle appears to have no

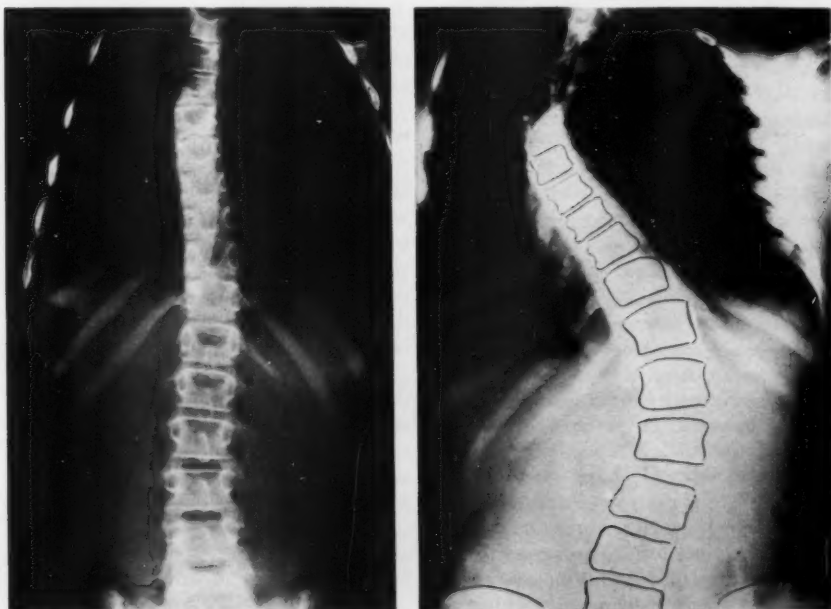


Fig. 9 — Type IV and usual pattern of progression.

constant relationship to the development of this curve.

This type is of great significance because it tends to progress to severe structural changes in both the spine, as well as the thoracic cage. It is of cosmetic, as well as physiologic significance.

Treatment of this type is most difficult. Manual, or plaster cast stretching has only limited value. Head traction, as applied through the use of a corset with head traction attachments, or the use of a Milwaukee frame is of value. Very early spinal fusion should be considered in this type.

Treatment of Early Scoliosis

While we all recognize the fact that some degree of scoliosis might develop despite our best efforts, we usually console ourselves by thinking that the really disastrous curves developed because the patient was either unable to tolerate the necessary care, or refused to comply with our prescribed care. This may well be true in a few cases, but our failure to prevent the development of serious degrees of structural scoliosis can usually be summarized by admitting that we did "too little too late." Looking back over my own failures, I recognize many faults.

1. In many instances, the curve was not discovered early enough, or, if it was discovered, its significance was not completely appreciated. In some instances, the x-ray visualization of the spine was not high enough to visualize the insidious development of a high thoracic curve and all attention was focused on the correction of a dorsolumbar curve of far less significance.

2. In some instances, even though curves were recognized, the patients were not followed at close enough intervals so that irreversible changes developed between the periods of medical re-examination.

3. Repeatedly, I have found that specific treatment was not instituted early enough, even though a curve was recognized. In some of these instances, it would appear that I did not want to make the patient or his parents unhappy by demanding limitation of a desired

activity, or the wearing of uncomfortable and conspicuous corrective apparatus.

4. In other instances, I found that I did not spend enough time explaining the importance of a scoliosis to the patient and his parents. They were not adequately motivated to carry out the tedious home program.

5. In other instances, I feel that I did not exert enough effort to obtain the necessary apparatus for control or correction, or did not check the apparatus closely enough to be sure that it was fitted correctly and was doing the job for which it was designed.

The treatment of early scoliosis may be divided into four components:

1. Regional mobilization.
2. Muscle re-education.
3. Support of spine.
4. Limitation of activity.

Each component is equally important, and all must be carried out adequately for optimum results. The neglect of any one will minimize the effectiveness of the others.

A. Regional Mobilization. The first logical step in the correction of a scoliosis is the attempt to maintain or regain regional mobility. It is important to re-emphasize here that asymmetrical hypermobility is no less important than asymmetrical contracture. Unless symmetrical mobility is attained, optimum results cannot be expected from muscle re-education, the use of orthotic devices to support the trunk, or surgery to maintain alinement of the spine. While we have methods of stretching out contracted areas, we have no practical way of tightening those tissues on the side of the convexity of the curve. For this reason, hypermobility may actually be more important than contracture.

The ideal method of regional mobilization is the patient's own effort to regain and maintain spinal alinement. Unfortunately, the patient is rarely able to correct faulty lumbosacral deviations, mid-lumbar curves, and high thoracic curves. It is, however, occasionally possible in the low thoracic and dorsolumbar curves, particularly when the patient is fitted with appropriate trunk support which shifts his trunk in a way

that requires him to correct his own faulty posture when sitting or standing. Active posture correction is far more effective in the development of compensatory curves.

Manual mobilization, or stretching of contracted tissues on the side of the concavity of a curve is frequently utilized and extremely valuable if carried out correctly. Very specific and effective stretching technics have been developed for mobilizing the low dorsal, dorsolumbar, and high lumbar regions. Specific, but less effective technics have been developed to stretch out the lumbosacral angle. In my hands, manual stretching of the high dorsal region has been non-specific and ineffective. Stretching of the spine should be done with the active participation of the patient, if at all possible, and movement should be carried out in a diagonal or anterolateral plane. Stretching in the lateral plane alone does not stretch the contractures present in the posterolateral tissues and may accelerate rotation of the bodies of the vertebrae and angulation of the posterolateral rib cage. Regional mobilization must be done gently, but persistently, and many times during the day. Stretching done once, or even twice a day is of little or no value, and heavy, forcible stretching is not permitted. If stretching is to be of any value, it must be initiated very early in the development of the scoliosis. Obviously, it must be carried out before bony structural changes take place, if realignment is expected.

Regional mobilization of the spine can be carried out effectively by a plaster cast so fitted to the trunk that localized stretching of contracted tissues is obtained. Further correction of spinal alignment can be obtained by modifying the cast several times each week. The tolerance of the patient to continued pressure over bony prominences and around the rib cage determines the degree of mobilization possible by this method. As is true of manual mobilization, plaster wedging is most effective in correcting low dorsal, dorsolumbar, and high lumbar curves. It has limited value in correcting lumbosacral angle devia-

tions and has little value in high thoracic curves.

Intermittent spinal traction is of great value in regaining mobility and alignment of the spine. When the patient's upper extremities are strong, an overhead bar can be utilized to permit the patient's own body weight to stretch out his spine. With the hands grasping the overhead bar, the body can be suspended for short periods of time many times a day. The use of a head halter, such as the Sayre head sling, also can be utilized many times a day to stretch out the spine by the suspended body weight.

Regional mobilization of the spine must be very carefully done if we are to avoid its several dangers. As mentioned previously, lateral pressure against the rib cage may cause angulation of the ribs and accelerate rotation of the body of the vertebrae towards the side of the convexity of the curve. Perhaps the greatest danger in attempting to mobilize one region of the spine is that of aggravating curves in adjacent regions, or of developing hypermobility above or below the regions being stretched so that eventually more dangerous curves develop. To stretch a spine correctly and without danger, whether by manual or by plaster cast methods, requires great skill and understanding of the mechanics of the spine. Utilization of the regional mobilization of the spine as a home routine must not be considered unless the parents can be taught very specifically, can demonstrate obvious ability to carry out specific instructions, and can be repeatedly checked by the trained physician or therapist. Overhead traction, however, can usually be carried out effectively and safely as a home routine. However, traction can never be as valuable as specific regional mobilization because traction can never obtain the symmetrical mobility so necessary for maintenance of alignment.

B. Muscle Re-education. Changes in the normal strength and balance of muscles in the shoulder girdle, trunk, and pelvic girdle may either be the cause, or the result of spinal curvature. Muscle re-education designed to strengthen specific muscle groups whose weakness can

be shown to contribute to the scoliosis is of great value, but such exercises must be carried out very skillfully. They are of real value only when the underlying spine is sufficiently mobile to permit correction of alinement, and when the physician or therapist supervising the exercise routine can accurately determine what is happening to the spinal alinement during the exercise period. Rarely can these routines be done safely by the patient and parents as part of a home program. In my experience, group gymnastics do far more harm than good as they tend to further stretch the paraspinal tissues on the side of the convexity, accelerate vertebral wedging and rotation, or many cause the development of dangerous curves adjacent to the curve under treatment. Muscle re-education is essential to the care of scoliosis, but, in general, it must be stated that it can be prescribed only after very accurate analysis of muscle imbalance by the physician and must be carried out under direct supervision of the trained physician or therapist.

C. Support of Spine.

1. *Orthotic Devices.* Externally applied apparatus to the pelvis, trunk, and thorax in the form of corsets, back braces, rigid jackets, adjustable frames, and so forth, have long been utilized in the care of scoliosis. Without doubt, they are an important and necessary part of over-all care. They are utilized to achieve four distinct but interrelated objectives: (a) to support weakness in the abdominal and lower and middle back musculature and maintain normal alinement of the underlying spine; (b) to correct alinement of the spine by direct pressure against regions of convexity; (c) to promote active correction by shifting the trunk to the right or left and thus demanding that the patient correct his own spinal alinement in order to maintain upright sitting or standing positions; (d) to correct spinal alinement by the force of traction pressing downward against the pelvis and upward against the head.

At Warm Springs, the basic trunk support is provided by a Hoke corset. All basic corsets have an aluminum bar

placed across the top and bottom of the posterior vertical stays to add over-all rigidity to the corset. This corset is used in place of a formal back brace because it is more acceptable to the patient and can be fitted and modified more easily. To this corset can be added a great number of attachments to provide pressure against levels of convexity in the dorsolumbar region. The use of the "three-point pressure" corset has been mentioned in the care of the Type II dynamic pelvic obliquity. The basic Hoke corset can also be used as the base for traction attachments that reach upward toward the head to provide the distractive force between the head and the pelvis. In the mobile dorsolumbar curve of Type III the top of the corset can be shifted towards the concavity of the curve and thus require that the patient in sitting or standing position correct his own alinement. The corset with head traction attachment is designed primarily to permit the patient sitting and

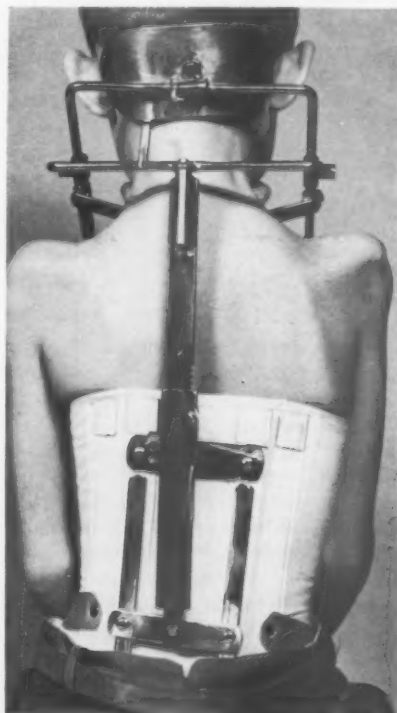


Fig. 10 — Corset with adjustable head traction.

standing activities while obtaining the corrective force of the traction. A Milwaukee frame replaces the corset during periods of rest, particularly at night. A rigid jacket of cellulose acetate or plastic material is not used except following spinal fusions. To this jacket may be added the traction attachment similar to that used on the corset.

While the importance of these devices is generally recognized, the limitations and dangers are not always equally appreciated. No corset alone has any practical effect in correcting deviations of the lumbosacral region, the mid-lumbar region, or in the upper thoracic region. When traction attachments are added to the corset, there may be significant control over these curves. However, the effect of traction, whether applied by attachments added to a corset

or built into the design of the Milwaukee frame, is limited in its value. At best, the patient can tolerate but a few pounds of traction when applied over a long period of time. In the sitting or standing positions, the weight of the head, upper extremities, and thorax far exceeds the amount of upward pressure that can be tolerated by the patient. Therefore, the effect of traction utilized in these positions cannot be compared to the effect when used in the lying position. Traction is also limited in that, at best, it can only correct and never over-correct. Over-correction is essential to ultimate complete control of any lateral deviation. A corset, even with all of its attachments, has very little effect on the patient who is obese. Obesity is one of the most difficult problems that we face in our attempts to control scoliosis.

The use of such devices may actually constitute a danger to the alignment of the spine. The use of a corset to support abdominal weakness might actually cover up the development of early deviations of the spine. Because the patient's back looks to be in good alignment when the corset is in place, the physician may neglect to examine the back critically when the corset is removed. However, the greatest danger in the use of such devices is in the development of rapid changes in the thoracic cage from regional pressure caused by the corset or back brace in its attempt to correct an underlying scoliosis. It has been long known, but not fully appreciated, that lateral pressure against the thoracic cage, particularly when the posterolateral ribs are angulated, causes an increase in their angulation and in the rotation of the bodies of the vertebrae towards the convexity of the curve. As emphasized many times previously, pressure against the rib cage on the side of the convexity should be against the posterolateral aspect of the ribs and not against the purely lateral regions. Actually, the ideal method of correction of rib cage deformity would be to pull out on the lateral aspect of the ribs rather than press in against them. It can probably be safely said that regardless of how well designed the apparatus is, if it presses



Fig. 11 — Modified Milwaukee frame.

against the rib cage at any point it is probably doing as much harm as good.

Corsets, back braces, and jackets also tend to limit chest expansion by encircling the middle and lower thoracic regions. This restriction of inspiratory excursion may be quite dangerous. A properly fitted corset should not extend higher than the xyphoid-sternal junction anteriorly and to a point just below the inferior vertebral angles of the scapulae posteriorly.

2. *Support Through Surgery.* Surgical procedures to maintain or correct alinement of the spine are not usually thought of as part of the early care of spinal deviation. However, it must be recognized that even surgery has little ability to correct structural changes in the vertebral column. Therefore, if there is an indication for surgery, it should be considered early and carried out, when at all possible, before significant structural changes take place. A thorough understanding of the significance of the four types mentioned previously permits us to recognize those curves that cannot usually be controlled by conservative means and demand the early consideration of surgical procedures. A typical Type IV curve, in my opinion, warrants very serious consideration of early spinal fusion. Hemiplegic and diagonal involvement of the abdominal wall is frequently controlled only by the use of fascial strips imbedded in the abdominal wall to counteract the force of the dynamic tension of muscles under voluntary and reflex control. It should be emphasized that if surgery is carried out, its success can be assured only if postoperative care is carried out consistent with the basic principles already mentioned and to be mentioned in this discussion.

D. *Limitation of Activity.* Intelligent control of the patient's activities is perhaps the most important and, at the same time, most frequently neglected aspect of the care of scoliosis. Positions of the spine during periods of rest can have marked influence on the spinal alinement. However, restriction of activity does not necessarily refer to positions of the body while at rest. Far

more important are those positions assumed during periods of sitting, standing, and moving about. In these positions, the weight of the head, upper extremities, and thorax exert great pressures downward against the spine. There is no type of orthotic device, or surgical procedure, that will fully protect the growing child with either severe diffuse, or severe asymmetric trunk weakness from developing spinal deviations if activity is completely uncontrolled. Frequently, the spinal alinement is endangered by over-enthusiastic attempts to increase the patient's functional capacity or ability to carry out the so-called activities of daily living. Very frequently, specific functional training must be markedly limited until spinal alinement is under control. Home instructions to the patient and his parents should include not only the time that the patient is permitted to sit, stand, and move about, but also those activities that are safe when he is in weight-bearing positions.

Conclusion

There is little doubt that scoliosis is the most difficult musculoskeletal deformity that we have to care for in the patient who has not reached mature skeletal growth. Unless persistent deviations of the spine are recognized early, and treated intelligently and persistently throughout all the growing years, we can never hope to control the alinement of the spine and to prevent significant structural changes. There are no "short cuts" to the care of this deformity.

References

1. Bennett, R. L.: Classification and Treatment of Early Lateral Deviations of the Spine Following Acute Anterior Poliomyelitis. *Arch. Phys. Med.* 36:9 (Jan.) 1955.
2. Cobb, J. R.: Outline for the Study of Scoliosis. *A.A.O.S.* 5:261, 1948.
3. Cobb, J. R.: Scoliosis; Quo Vadis? (Editorial) *J. Bone & Joint. Surg.* 40-A:507 (June) 1958.
4. Colonna, P. C., and vom Saal, F.: Study of Paralytic Scoliosis. *J. Bone & Joint Surg.* 23:335 (April) 1941.
5. Crow, Harold E.: Scoliosis. *A.A.O.S.* 5:236, 1948.
6. Farkas, Aladar: Paralytic Scoliosis. *J. Bone & Joint Surg.* 25:581 (July) 1943.

7. Ferguson, Albert B.: Roentgen Interpretations and Decisions in Scoliosis. A.A.O.S. 7:160, 1950.
8. Irwin, C. E.: Paralytic Scoliosis. A.A.O.S. 5:221, 1948.
9. James, J. I. P.: Paralytic Scoliosis. J. Bone & Joint Surg. 38-B:660 (Aug.) 1956.
10. James, J. I. P.: Idiopathic Scoliosis. J. Bone & Joint Surg. 36-B:36 (Feb.) 1954.
11. Kleinberg, Samuel: A Survey of Structural Scoliosis. A.A.O.S. 7:130, 1950.
12. Kleinberg, S.: Scoliosis: Pathology, Etiology, and Treatment, Baltimore, Williams & Wilkins Company, 1951, pp. 270.
13. Lowman, C. L.: Relation of Abdominal Muscles to Paralytic Scoliosis. J. Bone & Joint Surg. 14:763 (Oct.) 1932.
14. Moe, J. H.: The Management of Idiopathic Scoliosis, Clinical Orthopedics, Philadelphia, J. B. Lippincott Company, 1957, Vol. 9, pp. 169-184.
15. Rancy, R. B., and Shands, A. R., Jr.: The Prevention of Deformity in Childhood, Elyria, O., National Society for Crippled Children, Inc., 1941, pp. 168-175.
16. Reith, P. L.; Hopkins, W. A., and Dunlap, E. B.: A New Surgical Procedure in Scoliosis Therapy. Southern Surgeon 16:368 (April) 1950.
17. Risser, Joseph C.: Important Practical Facts in the Treatment of Scoliosis. A.A.O.S. 5:248, 1948.
18. Roaf, R.: Paralytic Scoliosis. J. Bone & Joint Surg. 38-B:640 (Aug.) 1956.
19. Steindler, Arthur: Nature and Course of Idiopathic Scoliosis, A.A.O.S. 7:150, 1950.
20. Willis, Theodore A.: Man's Back, Springfield, Ill., Charles C Thomas, Publisher, 1953, pp. 129-137.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



The Declaration of Independence places squarely on the shoulders of the individual the full responsibility, the full burden of his destiny, and not only of his own personal destiny, but of his common destiny with all other men in its ultimate fulfillment.

— R. L. BRUCKBERGER

Mechanical Properties and Temperature of Intact Skeletal Muscle in Patients with Muscular Dystrophy

Stella Y. Botelho, M.D.
Eleanor Bendler, M.D.
and
Sibyl B. Beckett, M.S.
Philadelphia

● The authors have reported previously that stimulation of the ulnar nerve produces abnormal mechanical responses of intact thenar muscles in patients with pseudohypertrophic muscular dystrophy. We subsequently have measured muscle temperature and found that a low muscle temperature cannot be entirely responsible for the abnormal mechanical responses for the following reasons: (1) thenar muscle temperature in the patients with pseudohypertrophic muscular dystrophy was not abnormally low; (2) about the same decrease in temperature occurred during a period of one hour in the patients and in the normal subjects, and (3) in normal subjects, abnormal mechanical responses were not observed in muscles which had been rendered ischemic although there was a significant decrease in temperature. Analysis of the mechanical properties of thenar muscles of muscular dystrophy patients and normal subjects of varying ages has produced two observations, which suggest that the dystrophic process in the adult type may differ from that in the childhood type: (1) intact thenar muscles of adult dystrophics were as stiff whereas those of childhood dystrophics were less stiff than normal, and (2) when compared to normal subjects, potentiation of single twitch tension after a conditioning tetanus was of lesser degree, shorter duration and was even replaced by depression in the childhood but not in the adult type of muscular dystrophy.

The characteristics of the electrical and mechanical responses of the indirectly stimulated intact thenar adductors of patients with pseudohypertrophic muscular dystrophy have been presented in detail elsewhere.¹ The abnormal findings can be summarized as follows:

1. A decrease in isometric tension upon induced thenar adduction was demonstrated even in patients who were still in the walking stage of this disease.

2. The objectively measured skeletal muscle weakness apparently resulted not only from a decrease in the number of muscle fibers but also from an inherent inability of the contractile tissue to generate tension.

3. The elastic properties of the intact muscle (contractile and/or non-contractile tissue) were changed such that the muscle was less stiff than normal. In the present study we have been able to demonstrate that the changes in the mechanical responses of intact skeletal muscle in patients with pseudohypertrophic muscular dystrophy could not be accounted for by abnormal muscle tem-

perature. In addition the data presented in the present paper indicate that not only do certain differences in skeletal muscle function exist between patients with the adult form of muscular dystrophy and normal adult subjects but also between patients with the adult and childhood forms of muscular dystrophy.

Methods and Subjects

The methods and apparatus (fig. 1) used in the present study have been presented in detail elsewhere.² In brief, the ulnar nerve was stimulated percutaneously at the wrist with supramaximal square electrical pulses. The electrical activity (action potential) and mechanical activity (isometric tension) of the thenar muscles which accompanied the induced adduction of the thumb were recorded by photographing the screen of a double beam cathode ray oscilloscope. All measurements were made after the records were magnified 15 times by means of a microfilm reader. Details of the measurements and typical records have been presented in previous communications.^{1,3} Experiments were performed upon male patients and normal male subjects in the following groups:

1. Sixteen patients with pseudohypertrophic muscular dystrophy (childhood type) ages six to 25 years, average 13 years.
2. Sixteen normal subjects ages six to 23 years, average 13 years.
3. Five patients with fascioscapulo-humeral muscular dystrophy (adult

From the Graduate School of Medicine, University of Pennsylvania, Philadelphia.

Supported in part by a grant from the Muscular Dystrophy Associations of America.

Read at the 3rd International Congress of Physical Medicine, Session on Electromyography, Washington, D. C., August 22, 1960.

type) ages 34 to 57 years, average 42 years.

4. Five normal subjects ages 32 to 43 years, average 38 years.

In addition, intramuscular temperature was recorded by means of a thermistor (Yellow Springs Tele-thermometer) in six patients in the wheelchair stage of pseudohypertrophic muscular dystrophy (ages 10 to 16 years) and in five normal adult male subjects (ages 25 to 30 years).

Results

Intramuscular temperature was found to be $37.7^{\circ}\text{C} \pm \text{S.E. } .25^{\circ}\text{C}$. in the normal subjects and $35.2^{\circ}\text{C} \pm \text{S.E. } 0.36^{\circ}\text{C}$. in the patients. Over a period of one hour there was a decrease of $0.06^{\circ}\text{C} \pm \text{S.E. } 0.01^{\circ}\text{C}$. and $0.17^{\circ}\text{C} \pm 0.11^{\circ}\text{C}$. in the normal subjects and in the patients respectively. None of these differences between the two groups was statistically significant. When a 10 minute period of

tourniquet ischemia of the tested arm was produced in the normal subjects, there was a decrease in temperature of 2.4°C .

When the data from *patients with adult muscular dystrophy* were compared with data from normal subjects of the same ages, the following results were obtained:

1. There were no significant differences between the parameters of the mechanical activity induced by single and tetanic (30/sec.) stimuli (table 1). On the other hand, all the measurements of the induced action potential in the adult dystrophics were less than normal (table 1).

2. When the degree of adduction was altered by passive stretch, both passive and active tension-length diagrams were the same in the dystrophics as in the normal subjects (fig. 2).

Table 1: (Mean Values) Characteristics of Induced Mechanical and Electrical Responses of Thenar Adductors

Age Range, Years	Over 30 Years		Under 30 Years	
	Dystrophy 34-57	Normal 32-43	Dystrophy 6-25	Normal 6-23
Single Twitch Tension				
Latent Period, msec.	8.11	8.5	13.5*	8.4
Passive Tension, gm.	302	385	90*	258
Max. Active Tension, gm.	375	469	43*	261
Contraction Period, msec.	57	53	63	60
Relaxation Onset, msec.	15	7.0	20*	9.0
% Relaxation Period, msec.	93	84	172*	89
Max. Contraction Rate gm./msec.	18.9	19.7	7.3*	19.6
Single Action Potential				
Max./Threshold (Area)	284*	685	305*	512
Amplitude, mV	5.920*	12.552	3.957*	12.461
Duration, msec.	6.7	8.52	6.3	5.5
Area, mm. ²	20,312*	29,816	8,779*	31,131
Repetitive Single Responses				
Fifth action potential	91	97	94	108
First action potential				
Fifth twitch tension	93	99	80*	99.6
First twitch tension				
Tetanus (30/sec.)				
Tenth action potential	73	73	73	76
First action potential				
Max. Active Tension, gm.	3,040	3,312	604*	2,410
Passive Tension, gm.	333	347	97*	283
Plateau number	12	13	15.5	12
% Change in Tension	9.2	8.4	16	9.5
Relaxation onset, msec.	37	37	54*	30.8
% Relaxation Period, msec.	98	106	325*	112.8

*Values statistically different from normal.

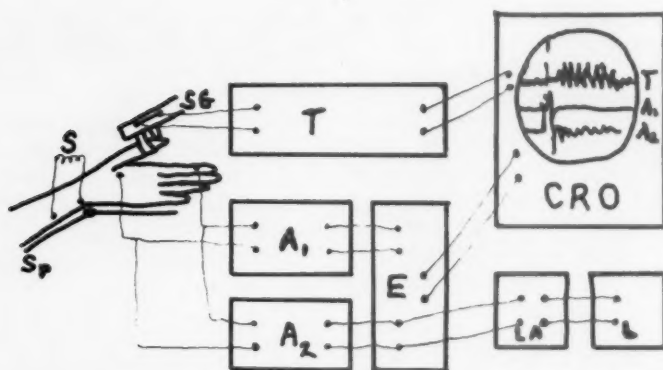


Fig. 1 — Diagram of the apparatus and experimental arrangements. The right hand was fixed to a splint (Sp), which could be adjusted at the wrist so that any degree of passive stretch of the thenar adductors could be maintained. Square electrical pulses, varying in strength from subthreshold to supramaximal, were applied to the ulnar nerve by means of a Grass S4B stimulator (S), Grass SIU4 isolation unit and surface electrodes placed at the wrist and forearm. Action potentials were led from silver disc surface electrodes (placed on the dorsum of the hand over the thenar muscles and the palmar surface of the distal phalanx of the index finger) through two Grass P5R AC preamplifiers ($A_1 + A_2$) in parallel, the input of a Dumont 330 electronic switch (E) to one beam of a Dumont 279 dual beam cathode ray oscilloscope (CRO) on which the potentials were displayed at both high (A_2) and low (A_1) gain simultaneously. The output of one preamplifier was also led through another amplifier (LA) to a loudspeaker (L). Mechanical activity was recorded as isometric tension by means of a strain gauge (SG), which was bonded to a bar, to which the thumb was attached. The input of the strain gauge was a 600 CPS current from a Sentinel Lab generator and the output was led, undemodulated through a Sentinel Lab amplifier (T) to the other beam of the cathode ray oscilloscope (CRO). The screen of the CRO was photographed on 35 mm. film, moving continuously at the rate of 1200 in./min. by means of a Dumont 321A oscillograph camera.

3. For a period of time after a tetanic stimulus (30/sec.) single twitch tension was greater than the control single twitch tension in normal subjects, a phenomenon which has been called post-tetanic potentiation of single twitch tension. Post-tetanic potentiation of single twitch tension was found to be of the same degree and duration in the dystrophics as in the normal subjects (fig. 3).

When the data from the *adult dystrophics* were compared with the data from the *childhood dystrophics*, certain differences were found:

1. When compared with normal subjects of the same age, not only was maximum tension of single twitches and tetanus reduced in the childhood dystrophics but the contraction kinetics were altered as indicated by the prolonged latent and relaxation periods and slow maximum contraction rate (table 1). These changes have been found to occur independently of changes in the simultaneously recorded induced action potential.¹

2. When the degree of adduction was increased by passive stretch in the

childhood dystrophics, the active tension-length curve was flat and the passive tension-length curve was less steep than in normal subjects of the same ages (fig. 2). The results of the present study confirm previous reports that older subjects have stiffer muscles than do younger subjects.⁴

3. Post-tetanic potentiation of single twitch tension was of less magnitude and duration and was even converted to depression in the childhood dystrophics (fig. 4).

Discussion

The decrease in active tension and prolongation of contraction and relaxation which occurred in the patients with pseudohypertrophic muscular dystrophy are similar to the changes which occur when a muscle is cooled. In the present study the muscle temperature was an average of 2.5 C. less than normal in the patients with pseudohypertrophic muscular dystrophy. However, we believe that this lower temperature cannot be entirely responsible for the marked changes in

tension and contraction kinetics for at least two reasons:

1. Tension and contraction kinetics of human thenar muscles are not altered when a temperature 2.4 C. less than normal is produced by tourniquet ischemia.³

2. Mammalian muscle temperature 6 C. less than normal must be attained before there is a decrease in tension of 22 per cent and over-all prolongation of twitch duration of 19 per cent.⁵

Based upon more detailed analysis of the data obtained from patients with pseudohypertrophic muscular dystrophy, we previously have reported that the decrease in active tension, the flat active tension-length curves and the altered contraction kinetics indicate that the skeletal muscle weakness results not only from a decrease in the number of con-

tractile units, but also from an inherent inability of the remaining contractile substance to generate tension.¹ The results of the present study indicate that, unlike the childhood dystrophic muscle, the indirectly stimulated thenar adductors of adult dystrophics generate a normal degree of tension and display normal contraction kinetics. This finding could be explained if: (1) the intrinsic hand muscles are involved in childhood but not in adult dystrophy or (2) if, unlike childhood dystrophy, there is loss of contractile substance in the adult dystrophic hand muscles but that the remaining contractile tissue has normal capabilities of generating tension.

In the present study we found that the induced action potential in the adult dystrophics was smaller and the ratio of maximal response to threshold response

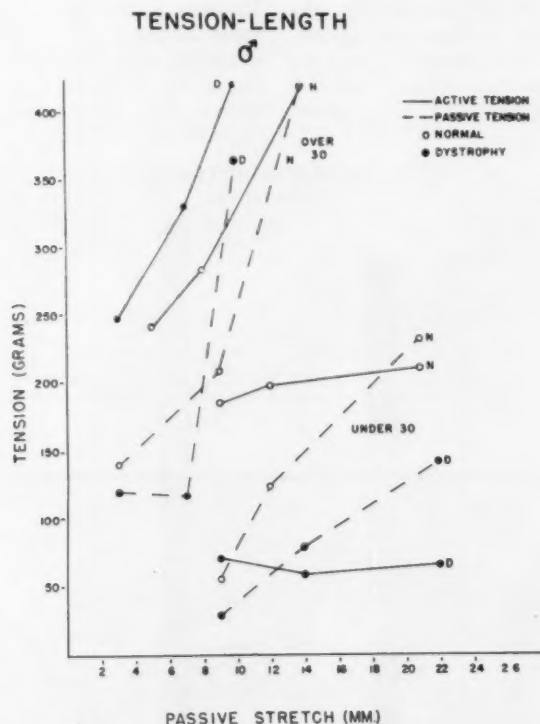


Fig. 2 — Tension-length diagrams. In this and succeeding figures, D and closed circles indicate the dystrophic patients, N and open circles indicate normal subjects; adult and childhood dystrophic patients are labelled over and under 30 respectively. Active tension is the maximum tension developed by thenar adduction which results from single supramaximal stimuli. Passive tension is the tension of the muscles at rest just prior to the single stimulus when there is no or very little electrical activity recorded with maximal amplification.

was less than normal. These observations indicate that there were fewer than normal muscle fibers responding to the maximal stimulus. We, therefore, conclude that although there is loss of contractile substance of the intrinsic hand muscles of adult dystrophic patients, the remaining contractile substance does have normal ability to generate tension.

From analysis of the passive tension-length curves, we have previously reported that the thenar adductors of childhood dystrophic patients are less stiff than normal. This finding is unlike that of muscles excised from genetic dystrophic mice.⁶ This observation could, therefore, be additional evidence to support the hypothesis that the disease process in human pseudohypertrophic dystrophy is somewhat different from that in genetic mouse dystrophy.⁷⁻⁹ This finding of decreased stiffness could be the result of changes in the elastic properties of either the contractile or non-contractile substance of the muscle. The work of Bourne and Golarz suggests

that the fundamental defect in human primary muscular dystrophy may be in the connective tissue.¹⁰ It is possible that studies of the elastic properties of the contractile and non-contractile substance of muscle would be useful in pseudohypertrophic muscular dystrophy to determine if this hypothesis is correct. Unlike the childhood dystrophic muscle, the thenar adductors of the adult dystrophic patients did not show abnormal stiffness, indicating that the elastic properties of these muscles were normal.

Another difference between the adult and childhood dystrophy was demonstrated when the effects of a given degree of activity, i.e. a conditioning tetanus of 30 sec. frequency for 1 sec., were studied. This degree of activity produced the same amount and duration of potentiation of the single twitch tension in the adult dystrophics as in the normal adults. However, in the childhood dystrophics a lesser degree and shorter duration of potentiation of twitch tension was produced by the same degree of activity. Indeed, this degree of activity produced

POST-TETANIC POTENTIATION OF OVER 30 YRS.

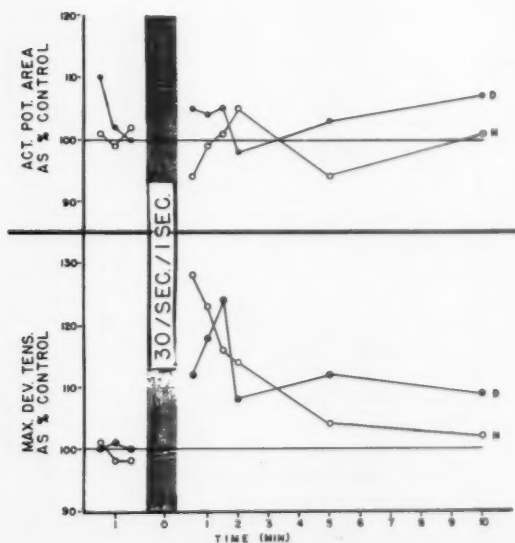


Fig. 3 — Post-tetanic potentiation in adult patients and subjects. The amplitude of the mechanical (maximum developed tension) and electrical (area of the action potential) responses are plotted in per cent of control values at varying intervals before and after a tetanic stimulation (30/sec. for 1 sec.).

abnormal depression of twitch tension in some of the childhood dystrophic patients. The exact cause of post-tetanic potentiation of twitch tension is not known. However, it has been shown both in mammalian¹¹ and human^{8, 12-14} skeletal muscles that post-tetanic potentiation is a function of the muscle and not of the neuromuscular junction, nerve or central connections. It is generally thought that post-tetanic potentiation of twitch tension unaccompanied by changes in the action potential such as is shown in the present study results from an altered chemical milieu in the recovery phase of muscle. This post-tetanic potentiation of twitch tension is to be differentiated from post-tetanic facilitation of neuromuscular transmission which occurs particularly in patients with myasthenia gravis. The latter is demonstrated by an increase in size of muscle action potential.¹⁵ The post-tetanic depression shown in the present study is not the same as post-tetanus exhaustion of neuromuscular

transmission which has been described as a decrease in size of the action potential after tetanus in patients with myasthenia gravis.¹⁶ The fact that a given degree of activity affects the childhood dystrophic muscle in a different manner from the normal and adult dystrophic muscle suggests that the biochemical events associated with the recovery of muscle after activity are altered in the childhood but not in the adult dystrophic muscle.

The differences between the mechanical responses of childhood dystrophic and adult dystrophic muscle which we have demonstrated in the present study indicate that the disease process is not identical in the two conditions. The difference could be merely a matter of distribution of the disease with regard to muscle involvement. However, we believe that the analysis of the electrical activity of the thenar adductors in the present study indicate that these muscles are involved both in the childhood and in the adult dystrophic patients. We,

POST-TETANIC POTENTIATION

♂ UNDER 30 YRS.

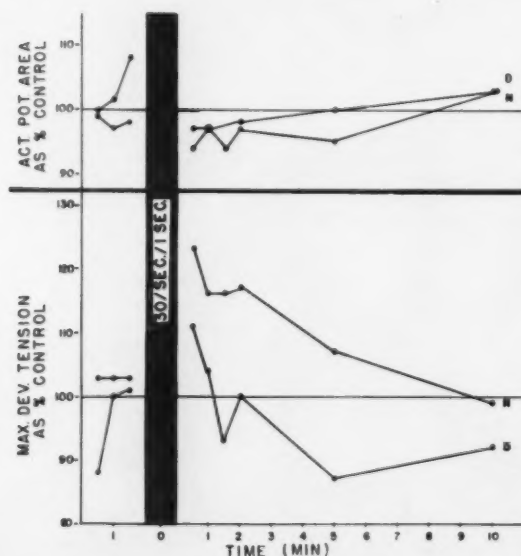


Fig. 4 — Post-tetanic potentiation in patients with childhood muscular dystrophy and in normal subjects of the same ages. The amplitude of the mechanical (maximum developed tension) and electrical (area of the action potential) responses are plotted in per cent of control values at varying intervals before and after tetanic stimulation (30/sec. for 1 sec.).

therefore, conclude that the fundamental disease process does differ in these two types of primary muscular dystrophy.

Summary

Analysis of the simultaneously recorded electrical activity and mechanical activity of the thenar adductors, which resulted from stimulation of the ulnar nerve, has been made. We have demonstrated that marked abnormalities in the amount of tension, the contraction kinetics, the elastic properties and the effects of activity occur in the childhood but not in the adult dystrophic patients. We have demonstrated changes in the electrical activity, which are consistent with a loss of contractile substance in the adult dystrophic muscle. For these reasons, we believe that the fundamental disease process differs in these two types of primary muscular dystrophy. In addition, we have demonstrated that the changes in the mechanical responses of the childhood dystrophic patients could not be accounted for by abnormally low muscle temperature.

References

1. Botelho, S. Y.; Beckett, S. B., and Bendler, E.: Characteristics of Mechanical and Electrical Responses of Intact Thenar Muscle to Indirect Stimuli: Study of Patients with Pseudohypertrophic Muscular Dystrophy. *Neurology* 10:601 (June) 1960.
2. Botelho, S. Y.: Alterations in Muscle Tension without Similar Changes in Electrical Activity in Patients with Myasthenia Gravis. *J. Clin. Invest.* 34:1403 (Sept.) 1955.
3. Botelho, S. Y., and Cander, L.: Post-tetanic Potentiation Before and During Ischemia in Intact Human Skeletal Muscle. *J. Appl. Physiol.* 6:221 (Oct.) 1953.
4. Botelho, S. Y.; Cander, L., and Guiti, N.: Passive and Active Tension-Length Diagrams of Intact Skeletal Muscle in Normal Women of Different Ages. *J. Appl. Physiol.* 7:93 (July) 1954.
5. Maclagan, J., and Zaimis, E.: The Effect of Muscle Temperature on Twitch and Tetanus in the Cat. *J. Physiol.* 137:89P (May 25) 1957.
6. Sandow, A., and Brust, M.: Contractility of Dystrophic Mouse Muscle. *Am. J. Physiol.* 194:557 (Sept.) 1958.
7. Baker, N.; Tubis, M., and Blahd, W. H.: Metabolic and Nutritional Studies in Mice with a Hereditary Myopathy. *Am. J. Physiol.* 193:525 (June) 1958.
8. McIntyre, A. R.; Bennett, A. L., and Brodkey, J. S.: Muscle Dystrophy in Mice of Bar Harbor Strain. *A.M.A. Arch. Neurol. & Psychiat.* 81:678 (June) 1959.
9. Oppenheimer, H.; deLuca, A., and Milhorat, A. T.: Serum and Plasma Proteins, Lipoproteins and Glycoproteins. III Hereditary Muscular Dystrophy in Mice. *Proc. Soc. Exper. Biol. & Med.* 100:568 (Mar.) 1959.
10. Bourne, G. H., and Golarz, M. N.: Human Muscular Dystrophy as Aberration of Connective Tissue. *Nature* 183:1741 (June 20) 1959.
11. Brown, G. L., and von Euler, U. S.: After Effects of Tetanus on Mammalian Muscle. *J. Physiol.* 93:39 (June 14) 1938.
12. Hughes, J. R., and Morrell, R. M.: Post-tetanic Changes in the Human Neuromuscular System. *J. Appl. Physiol.* 11:51 (July) 1957.
13. Hughes, J. R.: Post-tetanic Potentiation. *Physiol. Rev.* 38:91 (Jan.) 1958.
14. Hoffman, W. W.: Action of Veratrine on Human Skeletal Muscle. *Neurology* 8:917 (Dec.) 1958.
15. Johns, R. J.; Grob, D., and Harvey, A. M.: Studies in Neuromuscular Function: Effects of Nerve Stimulation in Normal Subjects and in Patients with Myasthenia Gravis. *Bull. Johns Hopkins Hosp.* 99:125 (Sept.) 1956.
16. Desmedt, J.: Nature of Defect of Neuromuscular Transmission in Myasthenic Patients: "Post-Tetanic Exhaustion." *Nature* 179:156 (Jan. 19) 1957.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



Reason is the mistress and queen of all things.

—CICERO

Analysis of Sounds from Normal and Pathologic Knee Joints

Herbert Fischer, M.D.
Chicago
and

Ernest W. Johnson, M.D.
Columbus, Ohio

● Sounds from knee joints passively moved in a standard manner were picked up with a sensitive microphone, recorded on a magnetic tape and later analyzed with a sonic analyzer and visually on an oscilloscope. Twenty-five normal adults, 25 patients with rheumatoid involvement of the knee and 25 patients with degenerative arthritis (osteoarthritis) of the knee were studied. The gross wave pattern, energy output and the frequency distribution were compared in the three groups. Normal knees produced a relatively regular pattern with the surrounding soft tissue exerting a damping effect which is constant in any one individual. In rheumatoid arthritis the entire pattern is distorted, while in degenerative joint disease, there are high amplitude spikes superimposed on a normal pattern. Other less striking differences are discussed. A review of the literature is included.

It has been observed for many years that moving diseased joints causes palpable crepitation. Since the knee joint is readily accessible and frequently involved early in rheumatoid arthritis and degenerative joint disease, we selected this joint for the analysis of joint sounds. We hoped that this study would provide information to differentiate rheumatoid arthritis and degenerative joint disease before other objective evidence was available.

Review of Literature

Auscultation, a method of examining the functions and conditions of the respiratory, circulatory, digestive and other systems by the sounds they produce, is one of the most ancient modes of diagnosis. From the classic passage in Hippocrates' work, *De Morbis*, "if you listen by applying the ear to the chest," we have evidence that direct auscultation was used more than 20 centuries ago.

Although the stethoscope was invented by Laennec in 1816 and in the following years was further developed by him,¹ it was not accepted by clinicians immediately. It was further modified and improved by Piory in 1928, and by C. J. B. Williams, Marsch, Bianchi, Bazzi and Baruch.

To our knowledge, the earliest reports of using auscultation of joints for diag-

nostic purposes were given, in 1885, by Hueter,² who used auscultation of joints for the purpose of localizing loose bodies in the knee joints by means of his Myo-dermato-osteophone. The first reports of auscultatory findings of joints in this country were made by Blodgett in 1902.³ He reported creaking, grating and cracking noises in joints, which he found to be most marked in patients with chronic arthritis.

In 1906, Ludloff⁴ found that sounds similar to those heard over the knees of patients with chronic arthritis could also be heard over the spine, sacrum and pelvis. Arthritis deformans was localized in the cervical spine and sacroiliac region by him using auscultatory methods. He described friction noises in the atlanto occipital region and in the posterior iliac spines. Ludloff was able to point out minimal arthritic changes in x-rays which were first reported as normal, after he had systematically listened to the entire spine, pelvis and sacral area.

Bircher⁵ used auscultation of the knees in 1915 as an aid in diagnosing meniscus lesions. In 1929, C. F. Walters⁶ reported on auscultatory findings of joints in 1600 unselected cases. He found an increase in intensity of sounds from 1.5 per cent in the first decade to 81.5 per cent in the eighth decade. He emphasized "finest grating is a sign of early arthritis," even with the absence of symptoms.

Joint noises were first presented graphically by Erb, in 1933.⁷ He used a contact microphone for recording.

An extensive study on joint noises with an attempt to localize surgical knee lesions

This study was partially supported by a grant from The National Institutes of Health, Rehabilitation Institute of Chicago.
Associate Professor and Director, Division of Physical Medicine and Rehabilitation, Department of Medicine, Ohio State University.

Read at the 3rd International Congress of Physical Medicine, Session on Arthritis, Washington, D. C., August 23, 1960.

was carried out by Steindler⁸ and published in 1937. After auscultating many joints with a conventional stethoscope, he later used a cardiophone connected to a crystal microphone which picked up joint noises via a short bell piece with a diaphragm. The electrical signal was amplified and connected, thereafter, to an oscilloscope from which oscillographic records were made. A high pass filter cutting out frequencies below 130 cycles per second, in order to dispose of the tremor of the holding hand, was used in the instrument adapted for Steindler's study. It was his conclusion that joint auscultation of the knees definitely was helpful, particularly in cases of injury to the semilunar cartilages. He also found that differences of diagnostic value could be detected in patients with rheumatoid arthritis and degenerative joint disease.

A series of 1500 cases was reported by Peylan in 1953.⁹ In the two years preceding his publication he had used direct auscultation of the knee joints. Recognizing the physiologic limitations of the human ear, Peylan also made use of highly sensitive electronic equipment for further investigation of the sound phenomena produced in normal and in pathologic joints. He found that simple auscultation of a joint could, at times, be the sole method of making a diagnosis; in many other cases it could be the first or easiest detectable finding suggesting a diagnosis which could be confirmed by other means.

The most recent publication on observations obtained by joint auscultation was in 1955 by Ekensten¹⁰ who did a comparative investigation on 400 large maxillary articulations by clinical, radiologic and micraphonometric methods. For his auscultations he used a crystal microphone with low frequency amplifier and an oscilloscope. He produced continuous recordings with the help of a camera, film and a motor to move the film. He concluded that sound recordings formed an integral part in the investigation of certain pathologic conditions in the maxillary articulation, especially in subluxations.

Procedure

Twenty-five student and staff physical therapists, from ages 20 to 54 years, were all questioned closely about any previous joint symptoms. Twenty-five patients with rheumatoid arthritis and 25 patients with degenerative joint disease, all of whom were either accessible in the Physical Medicine or Arthritis out-patient clinic, were selected. The diagnosis had been established by history, physical examination, x-rays, latex fixation and continued observation for periods up to 10 years. Mean age in the rheumatoid arthritis patients was 47.6 years and the mean duration of the disease was 11.3 years. In the osteoarthritis group the average age was 69 years and the joint complaints had existed for an average of 11.4 years.

Auscultation with a binaural stethoscope over the upper pole of the patella, in accordance with Peylan,⁹ was carried out first in three steps:

- (1) Slow extension . . 70-180° } 25 sec.
- (2) Slow flexion . . . 180-70° } \pm 10
- (3) Rapid flexion . . . \pm 0.5
- and extension . . . 1.0 sec. sec.

The speed of leg motion was determined by the speed which produced the maximum output of noise in each individual patient. After direct auscultation was completed and the optimal speed was determined, the joint noise was recorded on tape.

For sound pickup, an M-20 microphone with a 526-B power supply and a 165-A base was taped to the skin over the upper pole of the patella. The sensitivity of the microphone is given as 15 to 15,000 cycles per second. The sound emitted from the knees was originally recorded on a two-channel magnetic tape. The amplifiers for both channels were Viking-Full-Fidelity RP61.

For adaption of the microphone on the knee, a stethoscope rubber bell with a diameter of 1½ inches was used. This rubber bell adheres to the surface of the patella by suction. Before applying the rubber bell over the upper pole of the patella, the rim of the bell was coated with lubricant jelly to avoid friction noises between the skin and the rubber bell.

By using mild pressure, air was forced from the skin-bell-microphone air space, producing sufficient suction to keep the microphone in place. The pickup microphone was further secured by a one inch rubber band applied with mild tension around the knee (fig. 1).



Fig. 1 — Microphone with rubber bell and elastic band, ready for recording.

During the recording the person was sitting on an examining table with the legs suspended over the edge. The leg was moved by the examiner during the slow-rate recording, adapting the speed to maximum sound output. In fast motion the extremity was moved actively by the patient.

The knee noises were recorded on one channel; the pertinent information on the cases studied was recorded simultaneously on the second channel by the examiner.

Later it was found that one channel recording of both knee joint noises and commentary was sufficient. A calibrating signal of one millivolt peak to peak at 120 cycles per second also was recorded immediately before and after each joint noise recording (figs. 2, 3).

For evaluation of the relative noise output, the joint signal was recorded with a multichannel mirror galvanometer on photographic paper moving at a

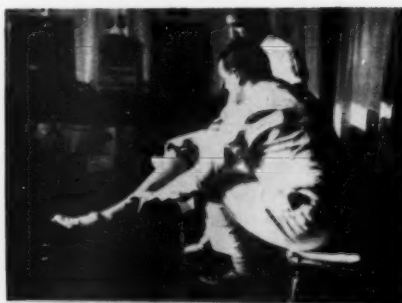


Fig. 2 — Recording in process. TR=tape recorder; OS=oscilloscope; AS=audio system; V=variable autotransformer; A= microphone preamplifier; P=calibrator and M=microphone attached to upper pole of patella.

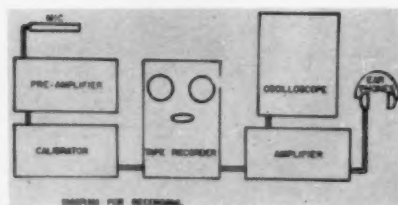


Fig. 3 — Schematic representation of recording equipment.

speed of approximately 1.1 centimeters per second. The use of two alternating charging capacitors, each with a charging time of one second, produces an integration of the joint noise signal. Different mirror galvanometers were used to record the joint noise, the integrated noise output, the identification of the patient and the position of the knee joint simultaneously.

Thus, a record with registration of the occurrence of sound at any particular position of the knee joint was obtained. At the same time, the amount of electrical activity was summated continuously and recorded at one-second intervals. A look at the tracing showed that the width of the areas covered by the integration pattern is kept constant. Therefore, the height in millimeters is proportional to the amount of noise recorded. The height values were added and then divided by the duration of the joint motion in seconds and by the height of the calibrator amplitude in millimeters. Thus, the average noise output in each

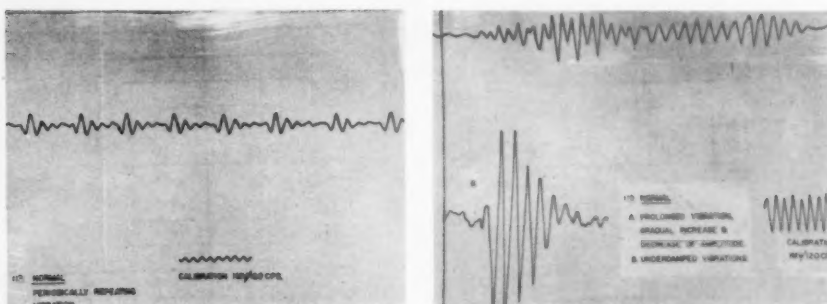


Fig. 4 — Recording of normal knees, paper speed 63.5 cm./sec., calibration signal 1 mv., 120 cps.

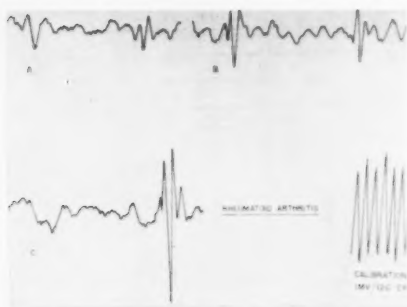


Fig. 5 — Recording of rheumatoid arthritis knee. (A & B), low amplitude grading and overdamped oscillations, (C), on left of tracing, overdamped oscillation with superimposed higher frequencies; high amplitude bursts on right of tracing, paper speed 63.5 cm./sec.; calibration signal 1 mv., 120 cps.

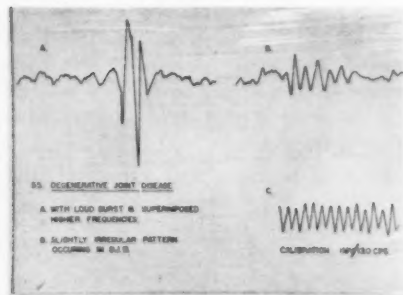


Fig. 6 — Recording in degenerative joint disease. (A), high amplitude burst with superimposed higher frequency oscillations, (B), slight alteration of "normal" pattern; paper speed 63.5 cm./sec., calibration 1 mv., 120 cps.

knee examined was determined (fig. 7). A reduction of the amplitude to comparable level was possible by the above-mentioned use of the calibration signal. For interpretation of the wave pattern, a

second recording with a fast paper speed of 25 inches per second (63.5 cm./sec.) was obtained. For this recording a Visicorder was used. All recordings were monitored with an oscilloscope and loudspeaker.

Damping of vibrations occurs in electrical and mechanical systems. Over-damped, critically damped and under-damped vibrations can be differentiated. Only in the under-damped cases do oscillations occur. These oscillations depend upon certain characteristics of the systems (in this case the knees), namely the mass, the friction and the elasticity. As it was observed on the scope, the vibrations appeared to have varying degrees of damping. The consequent analysis was to determine the degree to which these vibrations were damped. We hoped to determine the difference in damping between normal and pathologic knees, since alterations of friction and stiffness may occur in pathologic joints.

For computing the wave pattern obtained by fast writing speed, 63.5 centimeters a second, the equation

$$q = \frac{1}{1 + \omega_0^2 \times \tau^2} \quad (1)$$

where q is the damping factor, ω_0 is the observed angular frequency (of the damped oscillations) and τ is the time constant. However, when $\omega_0^2 \times \tau^2$ is very large (>625) then equation (1) can be approximated to

$$q = \frac{1}{\omega_0 \times \tau}, \text{ Stacy}^{11}$$

Frequency analysis was done with a vibration analyzer.

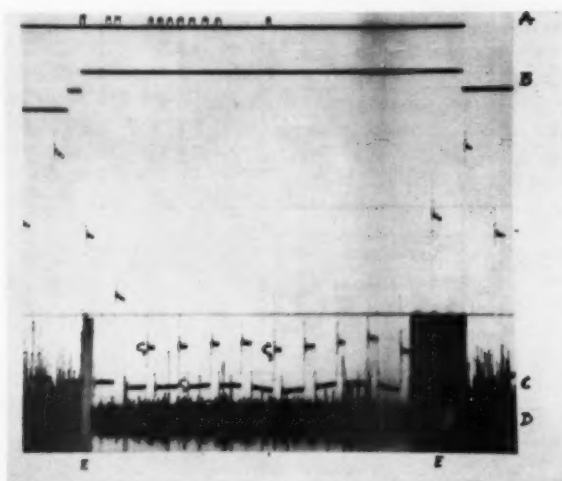


Fig. 7 — Recording for comparison of sound level. (A), identification marks; (B), indication of joint position; (C), integration signal; (C1), capacitor discharge; (C2), base line of integration signal; (D), joint noise recording; (E), paper speed 1.1 cm./sec., calibration signals 1 mv., 120 cps.

Results

The most characteristic findings in normal knees were the regularity in intensity and frequency of sound and the uniformity of the wave pattern. An average damping factor of 0.101 (table 2) and a frequency range from 10 to 300 cycles per second was found. The frequency increased slightly in higher age groups (fig. 9).

In rheumatoid arthritis patients, on direct auscultation, a sound was heard which is comparable to a pleuritic friction rub with a low frequency. The sound pattern is characterized by an irregular wave pattern due to an increase in the damping factor. This factor averaged 0.303 (table 2). Frequency of the sound was from 10 to 200 cycles per second with occasional bursts of higher frequency. Joint sound changes were noted before x-ray findings occurred in three patients with history of rheumatoid arthritis for three years (fig. 10).

In patients with degenerative joint disease, the most outstanding finding was the occurrence of high frequency, high intensity bursts which occurred at irregular intervals. Intervals between bursts were relatively quiet. The mean damping factor in degenerative joint diseased knees was 0.204; a frequency

range of 10 to 150 with bursts between 200 to 500 cycles per second was found (fig. 11).

Discussion

Frequently the characteristic sounds in normal and pathologic knees can be detected with a conventional binaural stethoscope. If one listens repeatedly to normal knees and knows the fine regular grating sounds produced on slow motion, it is easy to detect aberrations from this regular sound pattern. Based on direct auscultation, we thought that a comparison of the noise output in normal knees and in patients with rheumatoid arthritis and degenerative joint disease might prove helpful. We were surprised that the average noise output in patients with degenerative joint disease was less than in normals and in rheumatoid arthritis. In both normal and rheumatoid arthritic patterns, almost constant activity could be observed on the recording. In the patients with degenerative joint disease we observed high amplitude, high frequency bursts but, when integrated, these did not equal the constant low-level noise in the normals or the low-frequency thuds and gratings which are interspersed with an irregular pattern characteristic of rheumatoid arthritis.

Table 1: Comparison of X-Ray Findings and Auscultation in Early Rheumatoid Arthritis

Years of Duration	X-ray Findings	Joint Sound Abnormalities
2	N	N
3	N	+
3	N	+
4	N	+
4	+	+

X-ray and auscultatory findings in five patients with early rheumatoid arthritis. N = normal x-ray or auscultatory findings. + = abnormal x-ray or auscultatory findings.

Table 2: Summary Table Showing Average Noise Output, Frequency Analysis and Damping Factor

	R.A.	D.J.D.	Normals
Noise Output			
Slow Motion	26.39	19.40	26.93
Fast Motion	33.19	26.55	23.90
Frequency			
Analysis	10-200	10-150	10-300
Spikes	200-500	150-500	
Damping Factor ..	0.303	0.204	0.101

Average noise level, frequency analysis and damping factors in R.A. = patients with rheumatoid arthritis, D.J.D. = patients with degenerative joint disease, and normal knees.

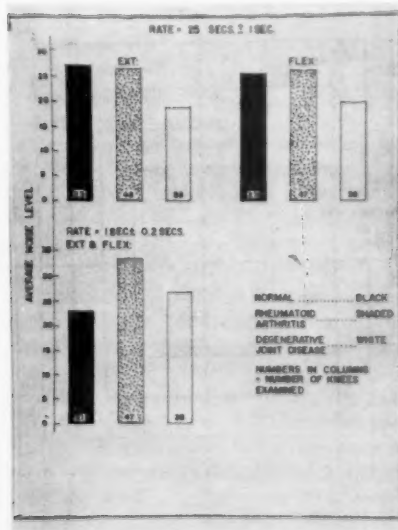


Fig. 8 — Graph: Average noise output on slow extension, slow flexion and rapid extension-flexion.

The calculations also showed that the rapid motion sound product in both rheumatoid arthritis and degenerative joint disease was higher per unit time than the sound produced during slow joint motion. In the normal knees the sound output on slow motion was more per unit time than on rapid motion.

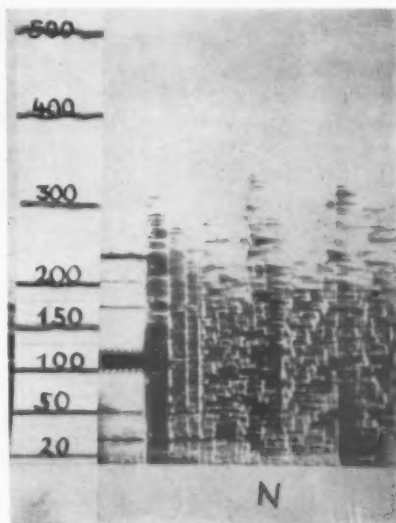


Fig. 9 — Frequency analysis of normal knee joint, calibration signals on left in cycles per second.

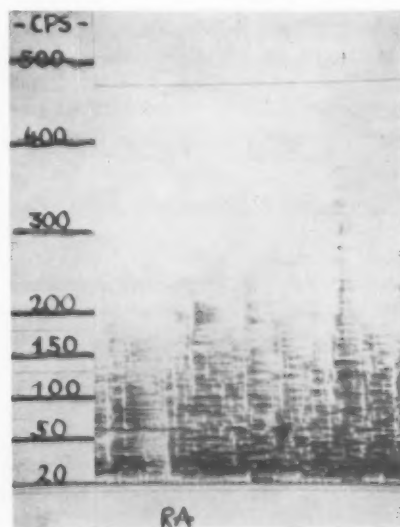


Fig. 10 — Frequency analysis in rheumatoid arthritis, calibration signals on left in cps.

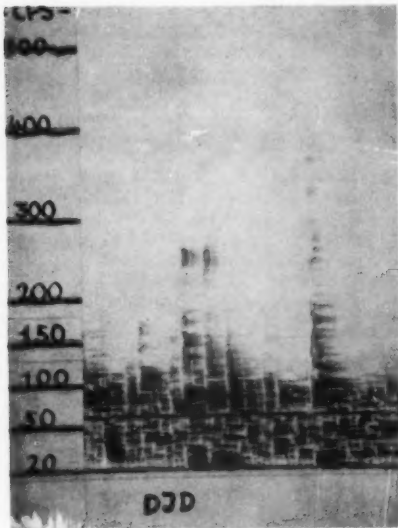


Fig. 11 — Frequency analysis in degenerative joint disease, calibration signals on left in cycles per second.

Comparing the sounds emitted from both knees in one person, we found that the sound product in normals was very little different. Sound production in flexion and extension was the same within narrow limits.

In pathologic knees, both rheumatoid arthritis and degenerative joint disease, the sound produced could be quite different from the opposite knee if the pathologic condition was unilateral or asymmetric in severity.

The wave patterns observed on the oscilloscope presented different degrees of damping. Since the damping is increased by friction, mass, intra-articular fluid and synovial thickening, we expected to find an increased damping factor in the pathologic knees. This assumption proved correct. It also explained, retrospectively, the relative lack of increase in joint sound in pathologic knees. Rougher joint surfaces undoubtedly produce more grating and cracking sounds than normals, but the increased damping in these knees prevents the occurrence of sustained sounds.

From inspection of the wave pattern it was obvious that, in the tracing of the rheumatoid arthritis recordings, we found both critically damped and over-damped

vibrations. Unfortunately, the equation used in our computations is not valuable in these instances. As computations made directly from the tracings are rather crude, further studies in which the tracings used for computations are enlarged photographically or by projection, to minimize the errors in measuring, are desirable. We attempted only to show trends of damping occurring in normal and pathologic knees.

The damping factor in degenerative joint disease is more than twice as much as in normals; in rheumatoid arthritis it is three times as large (table 2). These findings were expected since, in degenerative joint disease, the increased damping is produced by increased friction. In rheumatoid arthritis, however, the increased damping is produced by thickening of the synovial structures as well as by an increase in friction, especially in cases with long-standing disease. If this assumption is correct, then the damping factor may well parallel the pathologic course of the disease. The frequencies in table 2 are those occurring in the vibration patterns used when computing the damping factor. However, there are many frequencies superimposed on the basic pattern. The frequency analysis of the wave patterns did not reveal differences in any of the three groups of patients. The fast paper speed allowed more detailed analysis. We suspect that joint effusions may absorb the higher frequencies. This is another area for further investigation.

Conclusions

1. Development of refined electronic equipment makes detailed analysis of joint sounds a practical procedure.
2. A sensitive microphone, amplifier, tape recorder and an oscilloscope are the essential pieces of equipment for recording joint noises and allowing comparison with direct auscultatory findings.
3. Recognizable variations of sounds occur on passive and active motion of the knees in normal persons and patients with rheumatoid arthritis and degenerative joint disease. In rheumatoid arthritis these variations may occur before x-ray changes are evident.

4. Changes in the sound pattern observed in pathologic knees are probably due, in part, to an increase in the damping factor in these knees.

5. More investigation in this area is highly desirable to evaluate other joints and diseases as well as to make more quantitative studies.

References

1. Laennec, R. T. H.: *Traité de L'Auscultation Mediate et Des Maladies Des Poumons et du Coer*, 4th ed., 3 vol., Paris, J. S. Chaude, 1837.
2. Hueter, C.: *Grundriss der Chirurgie*, 3rd edition, Leipzig, F. C. W. Vogel, 1885.
3. Blodgett, W. E.: *Auscultation of the Knee Joint*. Boston Med. & Surg. J. 146:63, 1902.
4. Ludloff, K.: Die Auscultation der Wirbelsaeule, des Kreuzbeines und des Beckens. *Münchner Med. Wochenschrift*. 53:1197, 1906.
5. Bircher, E.: Zur Diagnose der Meniscus-luxationen und des Meniscusabrisse. *Zentralblatt fuer Chirurgie*. 40:1852, 1913.
6. Walters, C. F.: Value of Joint Auscultation. *Lancet*. 1:920 (May 4) 1929.
7. Erb, K. H.: Über die Möglichkeit der Registrierung Von Gelenkgerauschen. *Deutsche Ztschr. f. Chir.* 241:237, 1933.
8. Steindler, A.: Auscultation of Joints. *Bone & Joint Surg.* 19:121 (Jan.) 1937.
9. Peylan, A.: Direct Auscultation of Joints. *Rheumatism* 9:77 (Oct.) 1953.
10. Ekensten, B.: Jämförande Undersökningar av 400 st. Käkleder i Kliniskt, Röntgenologiskt och Fonogrammetriskt Hänseende. *Odontologist Tidskrift*. 63:18 (Feb. 28) 1955.
11. Stacy, R. W., and Malindzak, G. S., Jr.: Some Vibrational Characteristics of the Human Arterial System, to be published.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



He who will not reason, is a bigot;
 he who cannot, is a fool;
 and he who dares not, is a slave.

— WILLIAM DRUMMOND

Effect of Short Wave Diathermy on Radio-Sodium Clearance from the Knee Joint in the Normal and in Rheumatoid Arthritis

Dr. Ronald Harris
Buxton, England

● The radio-sodium clearance technic of Kety has been used to compare the effect of short wave diathermy on circulation in the normal and rheumatoid knees. In normal knees the increase in circulation averages 100 per cent. In quiescent rheumatoid knees there is a major increase averaging 60 per cent. In active rheumatoid disease major increases were not found, and in four out of five subjects there was a decrease in the circulation. This decrease is of the same order as found with intra-articular hydrocortisone and thus shows some rationale for using heat in treating active rheumatoid joints.

The rate of removal of radioactive sodium from a tissue has been shown by Kety¹ to be a quantitative measure of the local circulation. It has been applied to the study of the normal human knee joint.^{2,3} Simple exponential curves are consistently found, and from these, when the counting rate is plotted semi-logarithmically against time, straight lines are obtained, the slope of which gives the clearance constant K ,

(where $K = \frac{\log C_1 - \log C_2}{0.04343 (t_2 - t_1)}$, where C_1 and C_2 are counting rates at times t_1 and t_2 respectively).

For normal knees the clearance constant ranges between 0.020-0.090, with a mean value of 0.051.

Similar studies of the knee joint in patients with rheumatoid arthritis have been made.^{4,5} In these the clearance rate was found to vary widely. The range in rheumatoid knees with little or no local disease activity was the same as in normal control knees. In our series the clearance rate range was 0.032-0.090, with a mean value of 0.057. In knees showing severe local disease activity the clearance rate varied more widely and generally was higher, with a range of 0.048-0.191, and a mean value of 0.106. For subjects in whom the two knees were at different stages of disease activity, the sodium clearance was higher in the knee which was more severely involved. The effect of intra-articular injections of hydrocortisone was studied at intervals

from one to 14 days, and in general a reduction in clearance rate towards normal values was found. Marked clinical improvement in the joint was usually associated with a marked reduction in clearance rate towards the normal.

The effect of applying short wave diathermy to the knee joint in the normal and rheumatoid arthritic has similarly been studied.

Method

The technic used was that we have previously described.⁴ Between 0.2 and 0.5 ml. of isotonic saline, containing 5 to 10 microcuries of ²⁴Na, was injected into the resting knee joint, without local anesthesia. The counting rate was measured by ratemeters, connected to two unshielded scintillation counters arranged at either side of the knee joint. Plotting the pairs of readings, less the background count, semi-logarithmically against time gives a fairly straight line. After an initial period to allow stabilization of the curve, counting was carried on for 15 minutes, and then the knee heated for 20 minutes between the condenser electrodes of a standard short wave diathermy generator, tuned to maximum skin tolerance. The scintillation counters were removed from the diathermy field during the heating period. Counting was then resumed. (The ratemeters used were Type N522 Ekco electronics equipment, in conjunction with scintillation counters Type N509 Ekco electronics equipment. The short wave diathermy used was the Intertherm [Stanley Cox] with a frequency of 27.12 megacycles. A flexible rubber pad electrode size 18 x 12 cm., was used behind the knee; the other was

Consultant in Physical Medicine, Devonshire Royal Hospital, Buxton, England.

Read at the 3rd International Congress of Physical Medicine, Session on Arthritis, Washington, D. C., August 23, 1960.

Table 1: Effect of Heating the Normal Knee Joint with Short Wave Diathermy
Clearance Constant

	Preheating	Postheating	% Change
	0.023	0.070	
	0.028	0.050	
	0.033	0.052	
	0.034	0.055	
	0.034	0.084	
	0.044	0.092	
	0.046	0.120	
	0.079	0.109	
Mean	0.040	0.079	+97%

Table 2: Effect of Short Wave Diathermy on Quiescent Knees in Rheumatoid Arthritis — Stage 0
Clearance Constant

	Preheating	Postheating	% Change
	0.024	0.079	
	0.037	0.084	
	0.042	0.070	
	0.044	0.070	
	0.044	0.060	
	0.051	0.060	
	0.052	0.059	
	0.067	0.103	
Mean	0.046	0.073	+59%

Table 3: Effect of Short Wave Diathermy on Knees with Minor Activity in Rheumatoid Arthritis — Stage 1
Clearance Constant

	Preheating	Postheating	% Change
	0.032	0.059	
	0.035	0.037	
	0.045	0.077	
	0.046	0.070	
	0.078	0.110	
Mean	0.047	0.076	+62%

Table 4: Effect of Short Wave Diathermy on the Active Rheumatoid Knee — Stage 2
Clearance Constant

	Preheating	Postheating	% Change
	0.042	0.048	+14%
	0.069	0.062	-10%
	0.110	0.079	-28%
	0.114	0.059	-48%
	0.120	0.090	-25%
Mean	0.091	0.068	-25%

on an air spaced circular Bauwens type condenser electrode.)

The experiment was performed similarly for knees in patients with rheumatoid arthritis. The local activity of the knees was assessed clinically as follows:

0 = no involvement

1 = minor involvement

2 = major involvement.

(Group 2 was a composite of groups 2 and 3 of a previous investigation.⁴)

Results

Normal Knees. All eight subjects showed a major increase after heating, with an over-all increase of approximately 100 per cent. The mean initial value was 0.040, with a range from 0.023 to 0.079, and the postheating mean value was 0.079, with a range of 0.052 to 0.120.

Rheumatoid Knees. There were eight subjects with knees graded at stage 0, five subjects graded stage 1, and five subjects graded stage 2. The results for knees stages 0 and 1 were virtually identical with an over-all increase of 60 per cent:

Stage 0 — Mean preheating value 0.046; postheating 0.073.

Stage 1 — Mean preheating value 0.047; postheating 0.076.

Every subject showed an increase after heating.

The results for the active knees (stage 2) differed from the previous groups. Only one of the five subjects showed any increase, and this was small — 14 per cent. All the other four showed decreases, in one case as great as 48 per cent. Three of the five subjects had initial clearance values well outside the range of normal joint values (.110, .114, and .120) and these three all showed major reductions in clearance (28 per cent, 48 per cent, and 25 per cent respectively).

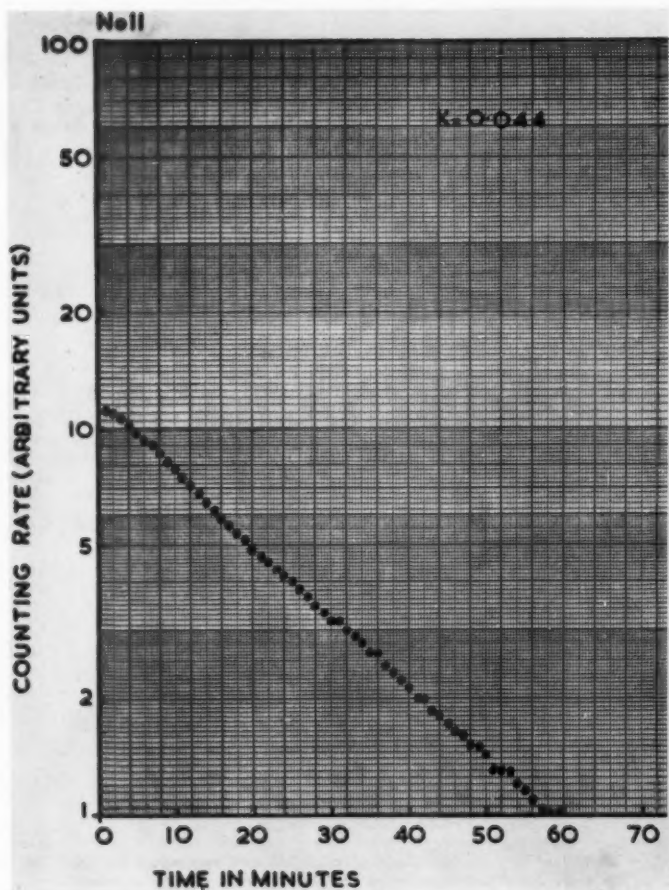


Fig. 1 — Sodium clearance curve from the knee joint of a normal subject, showing a straight line for 60 minutes. The gradient of the curve is a measure of the clearance rate (K = the clearance constant).

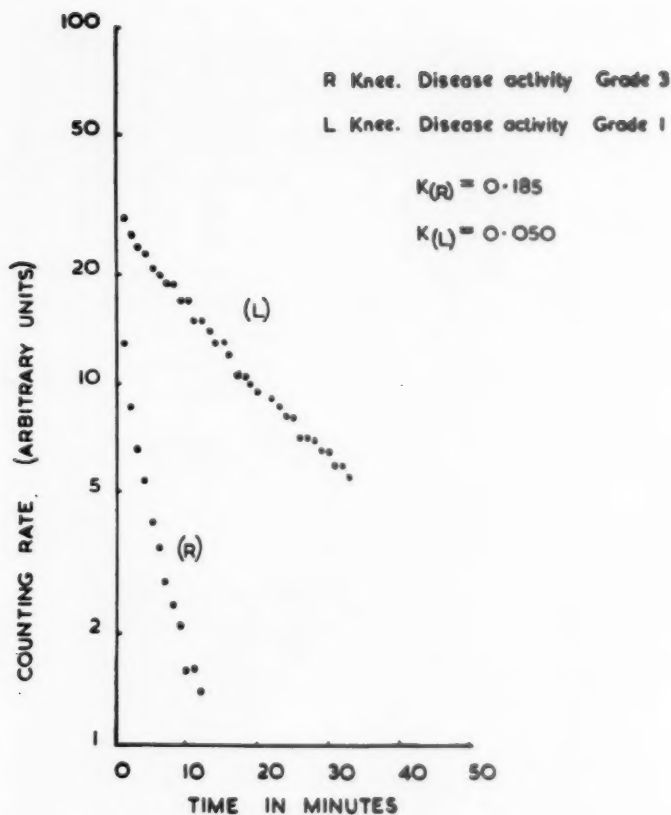


Fig. 2 — Sodium clearance from the knee joint of a subject with rheumatoid arthritis. The right knee has major local clinical activity, and the left knee has minor activity.

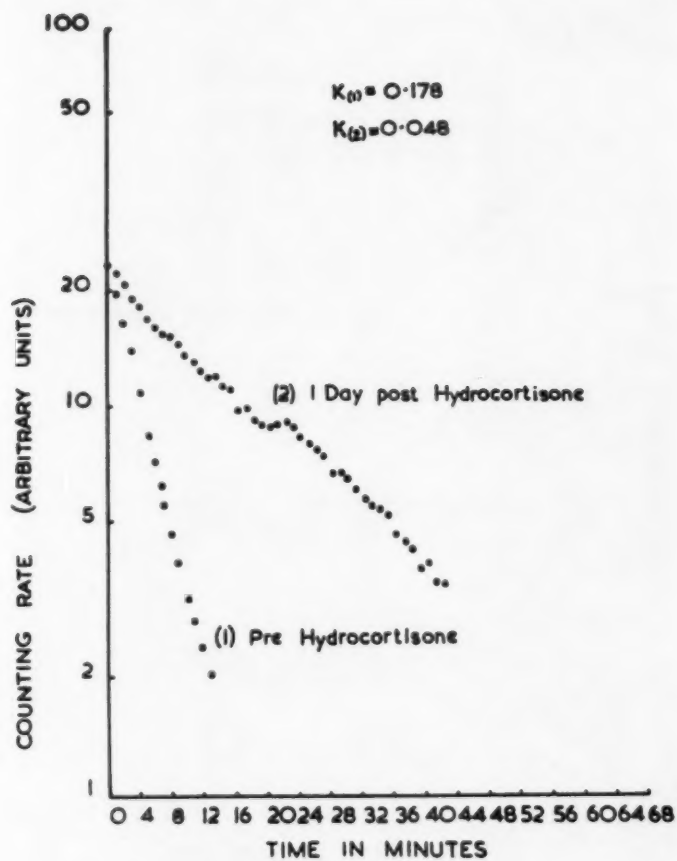


Fig. 3 — Sodium clearance curves from the knee joint of a patient with rheumatoid arthritis, before and 24 hours after intra-articular injection of hydrocortisone.

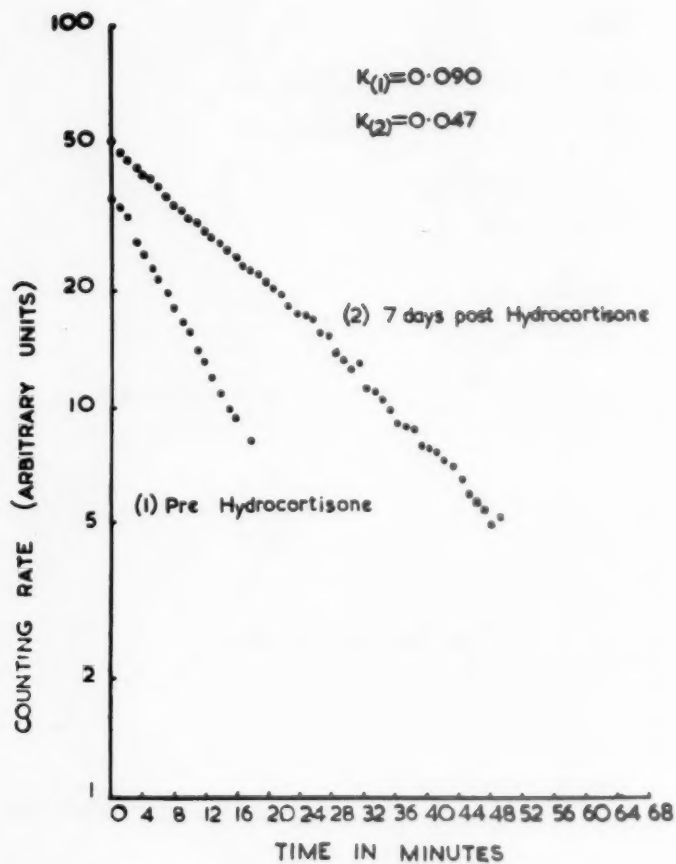


Fig. 4 — Sodium clearance curves from the knee joint of a patient with rheumatoid arthritis, before and seven days after intra-articular injection of hydrocortisone.

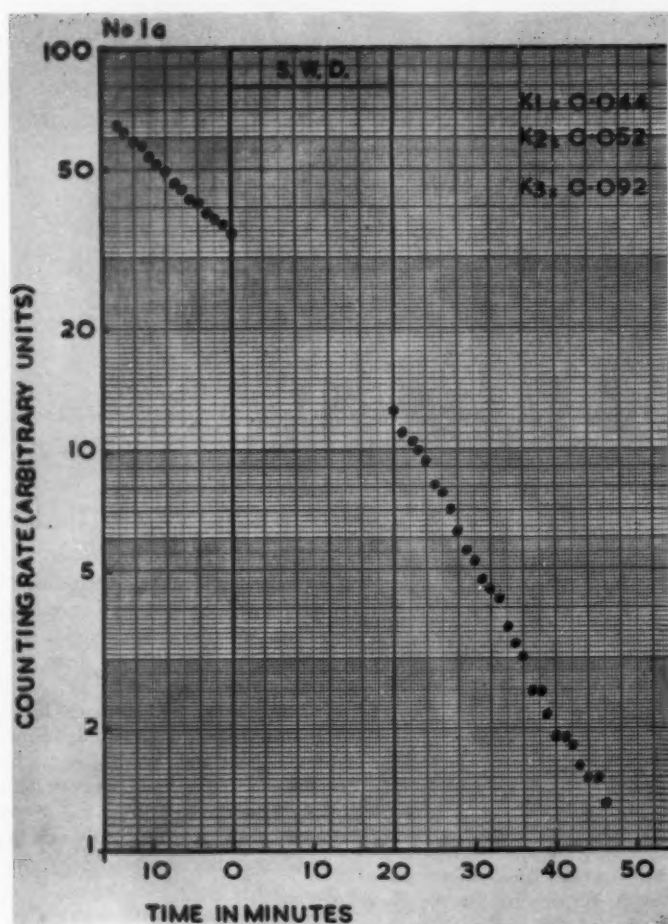


Fig. 5 — Effect of short wave diathermy on sodium clearance from the normal knee joint. A major increase in the gradient of the curve follows the heating. K_1 , K_2 , and K_3 are the clearance constants initially, during heating, and following heating, respectively.

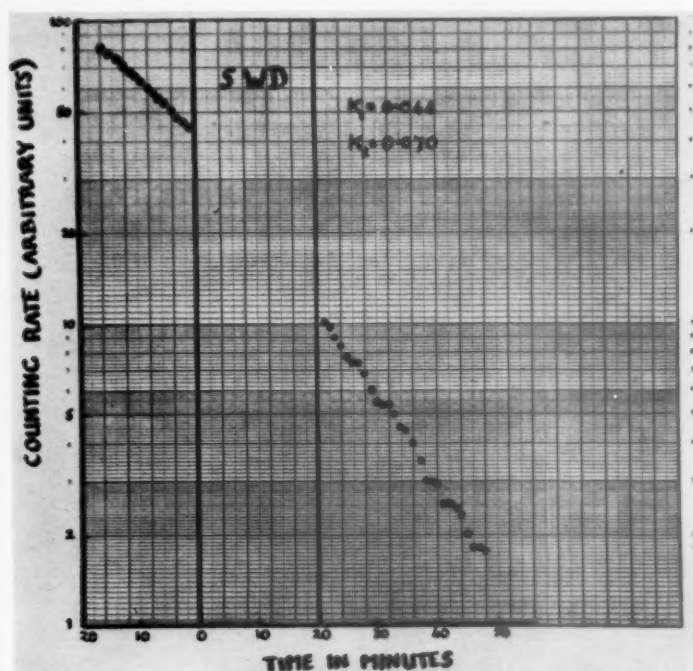


Fig. 6 — Effect of short wave diathermy on sodium clearance from the knee joint, in a patient with rheumatoid arthritis (minor local activity). A major increase in clearance follows the heating.

Discussion

The knee joints in which the rheumatoid process was not very active responded to diathermy in the same manner as did normal knees, that is with an increase in sodium clearance, indicating hyperaemia. Although initial values in the normal, the quiescent, and minor activity group were of the same order, and the post-heating values were not very dissimilar, the over-all results show that the proportionate increase in the normal group was decidedly greater than in the other two. However, the results for the knees with local activity differed considerably. Their initial value was high, showing a marked existing hyperaemia. The effect of heating the hyperaemic joint was not to further increase blood flow, but to reduce it sharply, back to within the normal range of clearance values. The findings are very similar to the effect of intra-articular hydrocortisone. In 13 subjects with active knee joints, intra-articular hydrocortisone produced a mean decrease in clearance of 24 per cent.⁴

Clinical improvement in the active rheumatoid joint is accompanied by a reduction in heat and swelling and a return of the hyperaemic synovia to normal. As applying heat might be expected to increase the temperature of the joint, and further increase the existing hyperaemia, it has seemed illogical to heat the active rheumatoid joint. However, our experiments show that local heating reduces the hyperaemia. This provides some rationale for using local heat therapy in rheumatoid arthritis.

Summary

The radio-sodium clearance technic of Kety¹ has been used to compare the effect of short wave diathermy on circulation in the normal and rheumatoid knees.

In normal knees the increase in circulation averages 100 per cent. In quiescent rheumatoid knees there is a major increase averaging 60 per cent.

In active rheumatoid disease major increases were not found, and in four

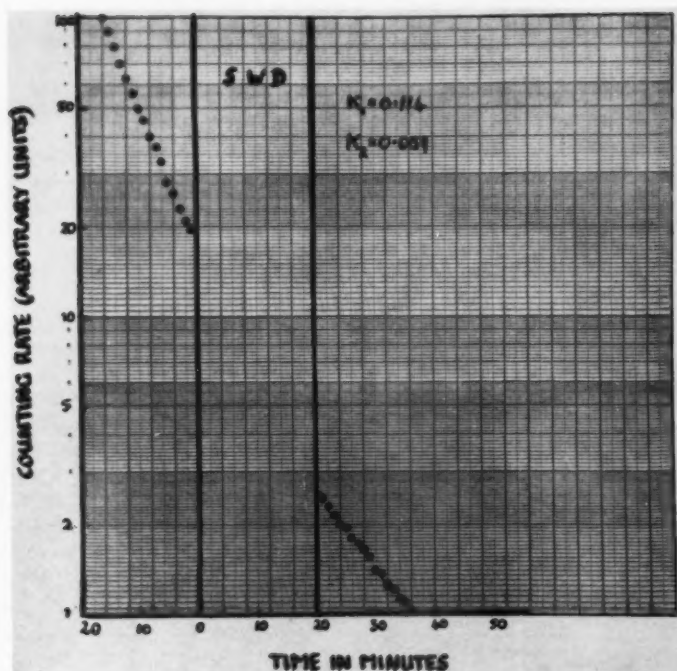


Fig. 7 — Effect of short wave diathermy on sodium clearance from the knee joint in a patient with rheumatoid arthritis (major local activity). A major decrease in clearance follows the heating.

out of five subjects there was a decrease in the circulation.

This decrease is of the same order as found with intra-articular hydrocortisone and thus shows some rationale for using heat in treating active rheumatoid joints.

References

1. Kety, S. S.: Measurement of Regional Circulation by Local Clearance of Radioactive Sodium. *Am. Heart. J.* 38:321 (Sept.) 1949.
2. Jacox, R. F.; Johnson, M. K., and Koontz, R.: Transport of Radioactive Sodium Across Synovial Membrane of Normal Human Subjects. *Proc. Soc. Exper. Biol. & Med.* 80:655 (Aug.-Sept.) 1952.
3. Harris, R., and Millard, J. B.: Clearance of Radioactive Sodium from Knee Joints. *Clin. Sc.* 15:9 (Feb.) 1956.
4. Harris, R.; Millard, J. B., and Banerjee, S. K.: Radiosodium Clearance from Knee Joint in Rheumatoid Arthritis. *Ann. Rheumat. Dis.* 17:189 (June) 1958.
5. Scholer, J. F.; Lee, P. R., and Polley, H. F.: Absorption of Heavy Water and Radioactive Sodium from Knee Joint of Normal Patients and Patients with Rheumatoid Arthritis. *Arthritis & Rheumat.* 2:426 (Oct.) 1959.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



Evaluation of Physical Disabilities by Means of Patient Profile Chart

O. Leonard Huddleston, M.D.

Richard W. Moore, M.D.

David Rubin, M.D.

Thomas L. Humphrey, B.S., R.P.T.

John W. Campbell, B.S., O.T.R.

and

Ron Blanchette

Santa Monica, Calif.

● For a long time a more effective and efficient method of evaluating physical disability and functional capacity has been needed in the field of physical medicine and rehabilitation. This paper presents a partial answer to this problem in the patient profile chart which was developed for this purpose. Values of muscle power and comparative functional capacities were arranged so that they could be recorded graphically for quick comparison and rapid evaluation. The functional activity scores and muscle grades are recorded by dots on the chart for the values measured, and subsequently connected from point to point to form a profile line. A muscle profile line is made for each side of the body and a single functional profile line is recorded for the functional tests. Subsequent profile lines are recorded at selected intervals and different colored profile lines are drawn for successive tests. In this way progress made by the patient may be compared easily by comparing the different colored profile lines. Also a quick comparison can be made between muscle power and functional capacity. The paper describes the clinical application of the patient profile chart and recommends that the chart be used as a research instrument in the future.

For a long time a more effective and efficient method of evaluating physical disability and functional capacity has been needed in the field of physical medicine and rehabilitation. A partial answer to this problem has been solved at the California Rehabilitation Center where the patient profile chart was developed for this purpose. Values of muscle power and comparative functional capacities were arranged so that they could be recorded graphically for quick comparison and rapid evaluation. Its clinical use was tested on a study of chronically disabled anterior poliomyelitis patients and traumatic quadriplegia patients. After many modifications, a suitable chart was worked out, which enabled us to record the data graphically at selected intervals, and ascertain the progressive changes during the recovery period.

Description of Patient Profile Chart

Figure 1 is an illustration of the patient profile chart. It consists of three evaluation forms, which were modified

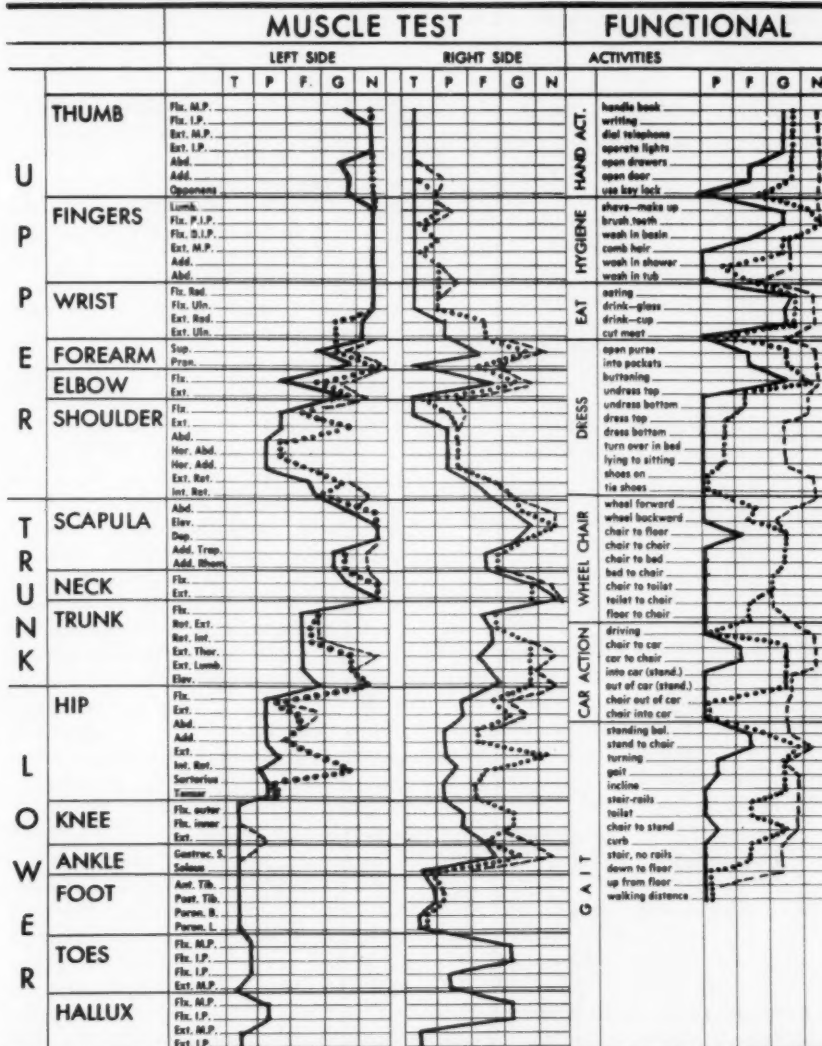
and combined into a one-page chart. The test forms used were the National Foundation for Infantile Paralysis Muscle Test,¹ and the two Functional Activity Test Records, used respectively in the Occupational Therapy and Physical Therapy Departments at the California Rehabilitation Center. Muscle power and functional activities were arranged to correspond to each other horizontally, as closely as possible, and the order of arrangement from top to bottom divided into three categories: (1) upper extremities, (2) trunk, and (3) lower extremities; these were further subdivided as follows: thumb, fingers, wrist, forearm, elbow, shoulder, scapula, neck, trunk, knee, ankle, foot, toes and hallux. The usual power grades for muscle testing were retained, namely, 0, zero; T, trace; P, poor, F, fair; G, good, and N, normal, with plus or minus for each of the large grades above T. The functional activities were arranged vertically from top to bottom as follows: hand activities, hygiene, eat, dress, wheelchair activities, car (automobile) activities and gait, with appropriate subheadings shown in the profile chart. The key for the functional gradings are shown at the lower right hand corner of the chart and consist of the following: P minus, attempts act; P, with assistance; P plus, requires little assistance; F minus, performed once; F, performs consistently; F plus, activity utilized; G minus, activity utilized in any situation; G, performs with ease; G plus, performs with ease in any situation, and N, no visible impediment.

From the California Rehabilitation Center, Santa Monica, Calif., and the University of Southern California School of Medicine, Los Angeles, Calif.

Read at the 3rd International Congress of Physical Medicine, Session on Medical Problems Diagnosed or Treated by Physical Medicine, Washington, D. C., August 25, 1960.

PATIENT PROFILE CHART

NAME J. W. CHART NO. 3252 DOCTOR RUBIN
 DIAGNOSIS POLIO AGE 37 ONSET NOV. 1955



MUSCLE TEST	1st Blue	2nd Red	3rd Green	4th Blue	5th Red	6th Green	7th Blue	8th Red
DATE	8/18/56	10/29/56	8/30/57					
THERAPIST								

FUNCTIONAL	1st Blue	2nd Red	3rd Green	4th Blue	5th Red	6th Green	7th Blue	8th Red
DATE	3/15/56	10/29/56	4/21/57					
THERAPIST								

COMMENTS KEY TO PROFILE LINES:

1st. _____
 2nd. ..-.....
 3rd. - - - - -

FUNCTIONAL TEST RATING

P — Attempts act
 P — With assistance
 P + Little assistance
 P — Performed once
 P — Performs consistently
 P + Act utilized
 G — Act utilized in any situation
 G — Performs with ease
 G + With ease any situation
 N — With little visible impediment
 N — No visible impediment

Fig. 1 — Patient Profile Chart of chronic anterior poliomyelitis patient showing profile lines of three successive evaluation tests. Differently constructed profile lines have been substituted for colored crayon profile lines which are customarily employed in clinical use of the chart.

Use of the Profile Chart

The functional activity scores and muscle grades are recorded by dots on the chart for the values measured, and subsequently connected from point to point to form a profile line. A muscle profile line is made for each side of the body and a single functional profile line is recorded for the functional tests. Subsequent profile lines are recorded at selected intervals and different colored profile lines are drawn for successive tests. In this way progress made by the patient may be compared easily by comparing the different colored profile lines. Also a quick comparison can be made between muscle power and functional capacity. These evaluation features are illustrated in figure 1.

Clinical Application of Patient Profile Chart

A study was conducted to ascertain in general the clinical uses of the profile chart and to determine what static and recovery trends might be found in a series of chronic "postpolio" patients. Analysis of the records revealed the following practical utilities of the profile chart:

1. Record a concise graphic progress report of the physical and functional status of the chronic "postpolio" patient.
2. Assist in formulating directives for conducting the total therapy (physical medicine and rehabilitation) program.
3. Facilitate the formulation of a prognosis regarding a more accurate prediction of the achievable recovery.

After an exhaustive preliminary search was completed of more than 2,000 charts of "polio" patients treated at the California Rehabilitation Center, it was possible to select a rather large number of patients with almost exact counterparts as far as the distribution of paralysis was concerned. A detailed study was conducted on 35 chronic postpoliomyelitis patients, using the patient profile chart. Most of the patients in each group received intensive resistive

exercise treatment for more than a year. It was possible to classify the patients into the following groups: Group 1, monoplegia; group 2, low paraplegia; group 2a, high paraplegia; group 3, triplegia; group 4, partial quadriplegia; group 4a, severe quadriplegia, and group 5, complete paralysis. The classifying characteristics of each group, together with their significant functional capabilities, chief causative physical disabilities, and their functional potentialities and capabilities made possible by treatment, including the use of assistive devices, are tabulated in table 1. Thus, it has been possible to obtain an objective picture of segmental functional activities of these respective groups.

In group 1, monoplegia, the functional deficiency is concerned largely with getting from one level to another without hand supports, namely, crutches or a handrail. The necessity of using the hands for elevation or ambulation interferes with many functional activities requiring the use of both hands; however, such an individual is not significantly limited in any essential self-care activities. Group 2, low paraplegia, shows that the presence of hip extensor power is of key importance, since these muscles usually insure a functional crutch and brace gait if the upper extremities are essentially normal. Absence of hip extensors interferes markedly with crutch ambulation. In group 2a, high paraplegia, the significant physical disability which differed from group 2 was that of paralysis of the trunk musculature. The trunk extensors are found to be much more important than the abdominals in propelling a wheelchair well. The loss of the trunk musculature of this group, at least for the first year after the onset of polio, also manifested itself in difficulty with transferring activities. It was observed, however, that with sufficient recovery, time, and adequate treatment and training, no significant functional difference existed between the two groups. In group 3, triplegia, the typical triplegic depends essentially on one normal upper extremity for all functions. A long period of training is therefore necessary to obtain capabilities in some of the

Table 1: Classification and Description of a Group of Chronic Anterior Poliomyelitis Patients
Based on a Study of Patient Profile Charts

Group Number and Name	Classifying Characteristics	Chief Functional Disabilities	Chief Causative Physical Disabilities	Capabilities Physical Medicine and Rehabilitation
Group 1. Monoplegia ...	Normal upper extremities and one good lower extremity with the remaining lower extremity flail, including the gluteal musculature.	Getting from one level to another without hand support, curbs and stairs without rails.	Unstable knee and hip of one lower extremity.	Good ambulation with conventional crutches, short crutches and leg brace — fair ambulation with cane.
Group 2. Low Paraplegia	Normal upper extremities with functionally normal trunk, and flail lower extremities.	Elevation and ambulation.	Lack of hip extensors causes instability of pelvis and lower extremities in ambulation.	Good function as wheelchair patient. Good sitting balance. Ambulation with braces and crutches not very practical.
Group 2a. High Paraplegia	Normal upper extremities with functionally normal musculature (especially the erector spinae group) and flail lower extremities.	Elevation, ambulation. Some limitations in wheelchair activities (transferring activities) largely confined to wheelchair.	Weak trunk muscles, especially the trunk extensors (more than the abdominals).	Maintain sitting balance and ultimately able to perform transferring activities with time and training.
Group 3. Triplegia	One normal upper extremity and one flail upper extremity with severe paralysis of the trunk and lower extremities.	Elevation, ambulation, limitation of wheelchair activities. Difficulties in hygiene, feeding, dressing, bilateral hand functions.	Severe paralysis of trunk and three extremities.	With treatment and training, most activities can be learned to perform with one hand. Wheelchair ambulation with one arm drive. Difficulty in transferring activities.
Group 4. Partial Quadriplegia	Moderately severe to severe paralysis of both upper extremities, with severe paralysis of the trunk and lower extremities.	Elevation, ambulation, limitation of wheelchair activities, including transfer and propulsion. Limited activities of daily living (A.D.L.), dressing and undressing, in and out of wheelchair, on and off toilet, etc.	Weak scapular depressors and weak upper extremity muscles, especially shoulder extensors, elbow extensors and flexors. Inability to move back and forth in the seat of the wheelchair (lack of seating action).	With treatment and training, transfer activities may be learned. Wheelchair propulsion with lever or tender drive. Improved A.D.L. with assistive devices.
Group 4a. Severe Quadriplegia	One upper extremity flail and the remaining extremity with severe paralysis of the trunk and lower extremities.	Same as Group 4, plus further limitation of hand, wrist and forearm function (A.D.L.).	Severe paralysis of the trunk and four extremities.	Hand function improved with opposition splint. Baling improved with feeder. Limited wheelchair propulsion with one arm drive wheelchair with tender drive. Feeder improved with electric wheelchair.
Group 5. Complete paralysis ...	Essentially complete paralysis of all parts of the musculature of the limbs and trunk.	Same as Group 4a plus further reduction of A.D.L. Practically helpless.	Severe generalized paralysis — respiration use required part of the time.	Minimal A.D.L. with appropriate assistive devices.

essential physical functions, such as hand, hygiene, feeding and dressing activities. The development of dexterity and power, eventually obtained in the remaining upper extremity, provides good functional ability in most upper extremity maneuvers, including wheelchair ambulation with a one-armed-drive wheelchair. The most difficult problems encountered are those of transferring activities. With treatment and training, however, the essential transferring activities may be achieved in this group. Group 4, partial quadriplegia, showed the greatest variance in muscle power and functional activity. Patients with good wheelchair activities demonstrated that one of the most important groups of muscles used to perform all transferring activities, are the scapular muscles, especially the depressors. Weakness of the scapular depressors, accompanied by severe involvement of the trunk and lower extremities, excludes many transferring activities and makes the remaining ones extremely difficult. The reaching range also is markedly limited, not only because of weak shoulder muscles, but also because of the absence of trunk extensors. When the power of the trunk extensors is increased enough for the patient to lean forward without using his arms for support, he is able to greatly increase his effective range of reach. This is an important function for wheelchair patients in practically all activities. Severely involved quadriplegic "polio" patients with some shoulder adduction and trunk extension musculature can be rehabilitated sufficiently to propel their own wheelchair, by using specially designed wheelchair accessory equipment known as spring loaded wheelchair levers or fender drives.² These devices have made wheelchair propulsion possible for many patients in group 4 and even for some of the more severely involved quadriplegic patients. In group 4a, severe quadriplegia, the significant limiting physical functions in the activities of daily living were those of dressing and undressing, and of transferring activities, such as chair to toilet, chair to bed, or chair to car. A "scooting motion" back and forth in the seat of the wheelchair is a key motion for these

functional activities. This one motion, plus some limited upper extremity power, makes it possible for these functions to be accomplished. This was demonstrated clearly by three patients who were brought back to the Center for a short period of treatment and training, four years after the onset of "polio". The "scooting motion" was established by concentrated self-care training and heavy resistive exercises performed by the patient six or seven hours daily, for three to four months. The results achieved in all three patients were those of independence in performing the more important transferring functions and of improved wheelchair ambulation. The functional activities of this group were limited to hand functions (hygiene and feeding) and most of these were accomplished by the use of assistive devices. Two patients of this group accomplished wheelchair propulsion to a limited degree, using a one-arm drive with fender-drive attachment. One of these patients, and his wheelchair, is shown in figure 2.

This 32-year-old white male developed anterior poliomyelitis on Sept. 26, 1955. His diagnosis, on admission to the San Bernardino County General Hospital, was that of severe "polio quadriplegia," spinal, paralytic, acute stage. In addition to the paralysis of all four extremities and trunk, he had marked tightness and spasm, painful muscles of the neck, trunk, back, hamstrings, calf muscles and shoulders. He also had bowel and urinary incontinence, which lasted for five weeks. It was not necessary to put him in a respirator, but his respiratory muscles were weak. The patient was treated in the San Bernardino County Hospital for three and one-half months. On Jan. 28, 1957, he was admitted to the California Rehabilitation Center, where he received intensive treatment as an in-patient until July 3, 1957, when he was discharged to out-patient status. He was followed on an out-patient basis on a reduced treatment program until December, 1958, at which time he was discharged and returned to work. He was provided with an electric wheelchair in July, 1957. He subsequently was

trained to use a one-arm wheelchair, which assisted in further developing his upper extremity and his self-care activities, including some transfer activities and functions. He gained considerable power in his neck, trunk and right upper

extremity, with no significant return in his lowers.

In group 5, complete paralysis, only a minimal hand function was accomplished by appropriate mechanical assistive devices.

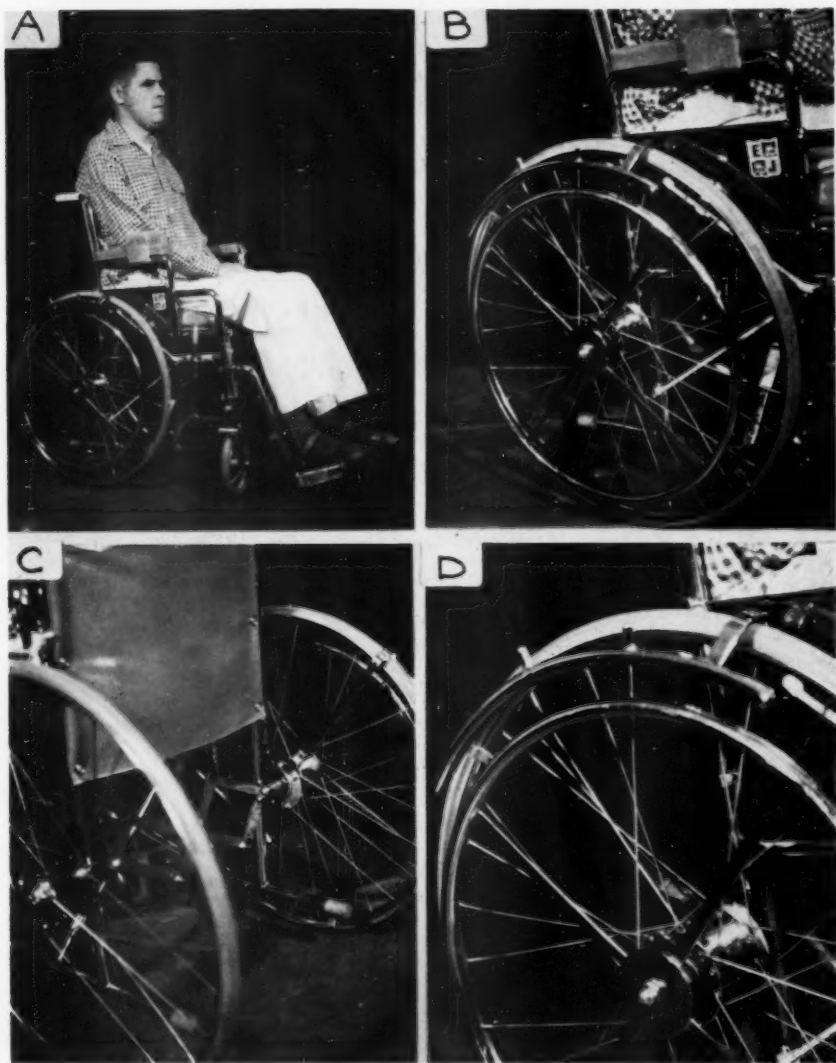


Fig. 2 — Chronic Anterior Poliomyelitis Patient, group 4a, severe quadriplegia, made vastly more functional by intensive physical treatment and a one-arm, drive-fender drive wheelchair: A. Patient seated in modified wheelchair showing fender drive and arm rest. B. Closeup of fender drive showing elastic attachments which position the fender when moved by propulsion. C. Showing flexible connecting mechanism for one-arm drive. D. Showing closeup of fender drive mechanism.

Conclusion of The Study on Chronic "Polio Patients"

Careful consideration of the profile charts of this group of patients provided the following general conclusions:

1. The results substantiated the belief that during the first six months following the onset of polio, the most rapid increase in muscle power takes place, and in many cases, the total recovery of muscle power is acquired.

2. The small increases in muscle power obtained after the first year of "post-polio" are the least significant in groups 1 and 2.

3. Small increases in muscle power obtained in groups 3 and 4, after one year, became increasingly important in proportion to the increase in involvement. With regard to the duration of treatment of severely paralyzed "polio" patients, the point of diminishing return is designated as that point at which increases in muscle power are too small to represent themselves functionally.

4. The point of diminishing return occurred sooner for groups 1 and 2 than for groups 3 and 4.

5. The rate of return of functional ability differed markedly from that of the restoration of muscle power. However, the rate was most rapid in groups 1 and 2 and progressed to the slowest in group 5.

6. During the first year of "postpolio," groups 3 through 5 showed an insignificant return of functional activity; in some cases, significant improvement was achieved in the second and third year or even later.

7. Patients with the greatest amount of recovery reached the point of diminishing return in the shortest period of time.

8. Patients with the least amount of recovery required a much longer period of time to reach the point of diminishing return.

9. The point of functional diminishing return was concurrent and occurred immediately after the point of muscle diminishing return in the patients with the greatest muscular recovery.

10. The more severely paralyzed the patient the greater the time required to

reach the functional point of diminishing return.

11. *Small increments in muscle power appeared to be increasingly significant in the more severely paralyzed patient.*

12. Classifying patients into groups according to segmental involvement allows for establishing more accurate prognosis with regard to their ultimate functional capabilities.

13. Segmental evaluation permits optimal therapy programs to be prescribed which can be directed more effectively toward improving physical motions and beneficial functions.

14. Even partial recovery of key regional muscle groups will greatly increase the functional capacity of chronic "polio" patients of groups 2 and 5 inclusive.

Profile Study in Traumatic Quadriplegia

A study, using the patient profile chart, was made on 50 traumatic quadriplegic patients who were in-patients at three different hospitals in the Los Angeles area. Sixty per cent of these patients showed injuries at the levels of the fifth and sixth cervical vertebrae and 40 per cent revealed injuries below the sixth cervical vertebra. Owing to the extremely diversified involvement of the patients with lesions below the sixth cervical vertebra they could not be classified into groups. A significant functional difference was found between the fifth and sixth cervical vertebrae. The latter group (C-6) had shoulder adductors, principally the sternal portion of the pectoralis major, and bilateral wrist extensors; these muscles were found to be the key power group which enabled the patients to perform transfer activities such as transferring from bed to wheelchair and wheelchair to toilet or automobile, whereas those with injuries at the level of the fifth cervical vertebra were unable to do so, because they were lacking in the above mentioned musculature.

Comment

One of the most important findings brought out by this study was that most of the test material used in physical

medicine is of such magnitude and relative importance that some method of handling the data easily and efficiently is necessary for use by the clinician and the research investigator. The patient profile chart enables one to recognize trends even at times where least expected to occur, and the simple organization of facts incorporated into a graphic profile chart helps in the recognition of these trends. Information regarding trends may be used to better direct the course of treatment of a given patient. Also the use of the profile chart is indicated in other areas of investigation.

Recommendations

Our study, extending over the past 10 years, showed that the average length of "polio" treatment at the California Rehabilitation Center for chronic poliomyelitis patients was one year. However, in view of the information revealed by the profile charts, it would appear that any fixed conception of the length of treatment is untenable. It is recommended, therefore, that a graduated length of stay would be preferable, perhaps six months or less for the least involved of group 1, one year to 18 months for groups 2 and 2a, and two years or more for the severely involved patient, groups 3 to 5 inclusive. Return visits of groups 2a to 5 inclusive to the Center once a year, for two or three months over a period of five years is advisable for the severely involved post-polio patients. Intensive manual resistive exercise therapy and functional training activities will provide in most instances, definite rewards in improved functional

activities. The treatment time would be spent in practicing the activities which need refinement and in attempting new activities, which heretofore seemed impossible. What appears impossible one year, may be readily achieved with intensive training on a subsequent return treatment interval the following year.

It is recommended that the patient profile chart be used as a research instrument in the future. A system of photographing and transferring pertinent information to punch cards, which subsequently can be analyzed with an IBM machine or computer, could conceivably reveal additional information of statistical significance which are not known at present. Such information may be used to establish standardized minimal muscle profile lines for patients with different types of physical disabilities. Likewise, standardized forms for predictable potentialities for the patients possessing given disabilities may be worked out on reference profile charts and the pertinent goals for treatment programs may be indicated. A system of graphic analysis and evaluation charts, based on patient profile lines, may assist in prescribing individual treatment programs to accomplish maximal rehabilitation recovery most effectively for disabled patients with given physical disabilities.

References

1. Daniels, L.; Williams, M., and Worthingham, C.: *Muscle Testing; Techniques of Manual Examination*, ed. 2, Philadelphia, W. B. Saunders Company, 1956.
2. Huddleston, O. L., and Campbell, J. W.: Wheelchair Fender Drive. *Arch. Phys. Med.* 39:314 (May) 1958.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



year, n.

a period of three hundred and sixty-five disappointments.

—Excerpt from the "Devil's Dictionary"
by AMBROSE BIERCE

Application of the Stretch and Hoffman Reflexes to the Objective Measurement of Spasticity

Oswaldo E. Miglietta, M.D.
and
Milton Lowenthal, M.D.
New York City

● Spasticity is encountered frequently in rehabilitation of patients with neuromuscular disabilities. The evaluation of spasticity and therapeutic procedures designed to control it remains a problem in clinical practice. Procedures previously described for objective evaluation of spasticity are either too elaborate for practical use or measure only in a qualitative manner the characteristics of the spastic muscle. We have approached the problem of evaluation of spasticity through a number of electromyographic parameters. This report describes experience with the stretch and Hoffman reflex in patients with various upper motor neuron disorders. The methods developed have been tested under clinical conditions and appear practical, readily reproducible and reliable. This presentation will describe the neurophysiologic factors involved, the technical features of the methods and their application in clinical settings.

In the rehabilitation of patients with upper motor neuron lesions, spasticity is one of the most difficult and often frustrating symptoms to control. Physical or pharmacologic measures are of limited value in control of spasticity. Surgical procedures such as neurectomy, posterior and anterior rhizotomy and cordectomy are at times resorted to in the most severe cases.

Equally as difficult as control is the problem of evaluating spasticity. The clinical criteria are well-known, and they range the gamut either of adjectives, such as "mild," "moderate," "severe" — or of numerical values, such as "one plus," "two plus," and "three plus." While these terms serve a useful clinical function they are inadequate when used to describe the therapeutic effects of procedures directed at reducing spasticity. This latter point is of particular concern in the field of Physical Medicine and Rehabilitation where the need for objective measurable criteria always is preferred over the clinical descriptive terms. In evaluating the effects of a therapeutic regimen and in assessing increment or decrement of function, objective criteria are the "conditio sine qua non."

The factor or factors responsible for the production of spasticity appear to be quite complex and still not adequately

identified. Spasticity is referred to as that abnormal state of muscle tone manifested by an increased resistance to passive manipulation of the limbs, hyperactive tendon reflexes and clonus. All these signs have a common denominator, namely an exaggeration of the stretch reflex.¹ The activity of the motor neuron is dependent on various afferent impulses reaching the anterior horn cells through many synaptic connections from the periphery and from higher centers in the central nervous system. The exaggeration of the stretch reflex in spasticity is apparently the result of impairment of central inhibitory influences, which normally reduce the activity of the anterior horn cells and of the unopposed activity of central facilitatory influences. The spastic state is further maintained by proprioceptive afferent impulses arising from the involved spastic muscles.

The purpose of this paper is to present two methods by which the activity of the anterior horn cells can be objectively and quantitatively assessed. These two methods comprise:

- a. measurement of the stretch reflex; and
- b. measurement of the maximum Hoffman reflex response.

Both methods make use of the reflex induced action potential response in muscles as a means to measure the excitability of the spinal motor neurons in spasticity.

Instructor, Department of Physical Medicine and Rehabilitation, New York Medical College.

Associate Professor, Department of Physical Medicine and Rehabilitation, New York Medical College.

This investigation was supported in part by a Fellowship from the Office of Vocational Rehabilitation.

Read at the 3rd International Congress of Physical Medicine, Session on Neuromuscular Diseases, Washington, D. C., August 24, 1960.

Objective Measurement of Stretch Reflex

In another paper² we are reporting in detail the technic used to elicit and measure the stretch reflex and the results obtained with a group of spasmolytic agents. The method is essentially based on the measurement of the maximal action potential amplitude picked up through skin electrodes from the muscle being stretched. The voltage amplitude of the reflex response, expressing the number of motor units reflexly activated, gives a measurable objective index of the reflex excitability of the spinal motor neurons.

Since the response of a muscle to stretch is dependent upon and proportional to the amount and rate of the stretch force,³ it is necessary to standardize the stretching force. This was achieved by use of an electrically activated hammer. This hammer was positioned at a fixed distance from the tendon; this assured that a constant blow was delivered.

The knee jerk was selected for study because the patellar tendon is easily accessible and the patient can be positioned comfortably on an elevated high seat chair (fig. 1). The ease of the testing procedure reduces the possible effects of discomfort and postural strain on the patient, and on the reflex activity of the spinal cord.

For recording the muscle action potential, several skin electrodes are simultaneously placed around the muscle belly thus permitting a rapid scanning of the muscle. The muscle scanning is a critical feature of the technic since the recorded response may vary in amplitude over the muscle belly, but a point is always found from which a maximal potential amplitude is obtained. This localized response is in accord with Lloyd's observation⁴ that the afferent impulse from a given extensor responding to stretch reflects into the muscle or the particular strip of the muscle being stretched, thus resulting in a very sharply restricted reaction.

The entire procedure is completed in 15 minutes. The time advantage is important when a large group of patients has to be studied and especially so in

light of the poor tolerance of patients with chronic disease to repeated and prolonged testing.

In figure 2 the response of the spastic and normal knee jerk of a hemiplegic patient is shown. The amplitude of the reflex response always is greater on the spastic side and can be used as a measure of the degree of spasticity. The amplitude recorded in a given patient is fairly constant and faithfully reproducible, with minimal variations, even after intervals of weeks and months.

The application of this measuring technic to the evaluation of spasmolytic

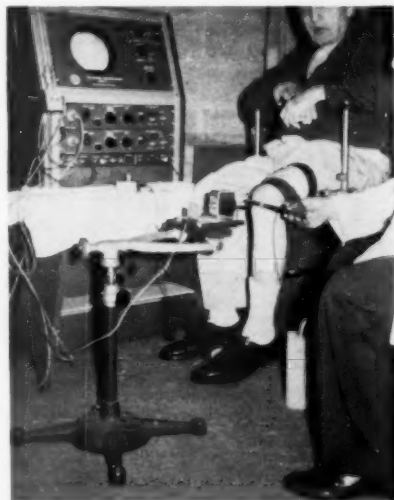


Fig. 1 — Set up for the elicitation of knee stretch reflex. The patient is comfortably seated on an elevated chair. The hammer is being placed at a fixed distance from the patellar tendon.

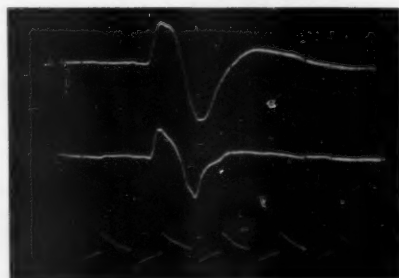


Fig. 2 — Action potential amplitude of a spastic (top line) and normal (second line) quadriceps muscle in a hemiplegic patient responding to stretch. Calibration: 1000 microvolts. Time: 30 milliseconds.

agents has been tested by us on a group of 62 spastic patients. Tables 1 and 2 show the amplitude of the reflex response before and three weeks after the patients were given different muscle relaxants. The shift to the left toward the lower values is evident from table 2 and indicates a decrease in the exaggerated reflex responses. The average amplitude decreased from 878 to 466 microvolts after three weeks of therapy.

Measurement of Maximum Hoffman Reflex

This reflex, introduced and described by Hoffman in 1922,⁵ is another method for a more direct appreciation of spinal cord reflex excitability. Hoffman demonstrated that submaximal electric shocks of the posterior tibial and femoral nerves in man evoked a reflex contraction of the gastrocnemius and quadriceps muscles

Table 1: Amplitude of Stretch Reflex in 62 Patients — Prior to Medication

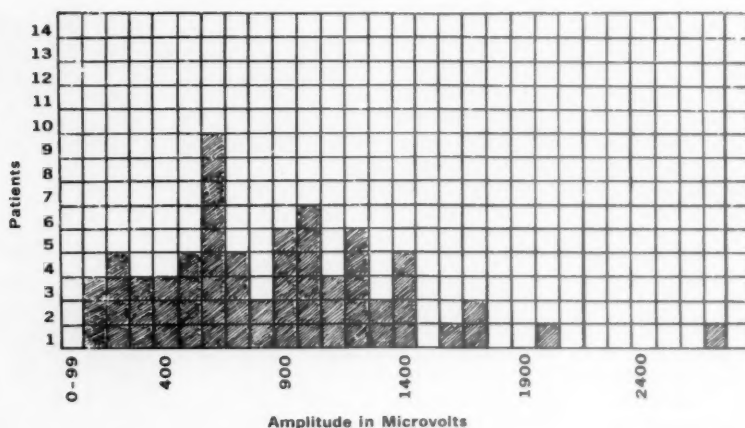
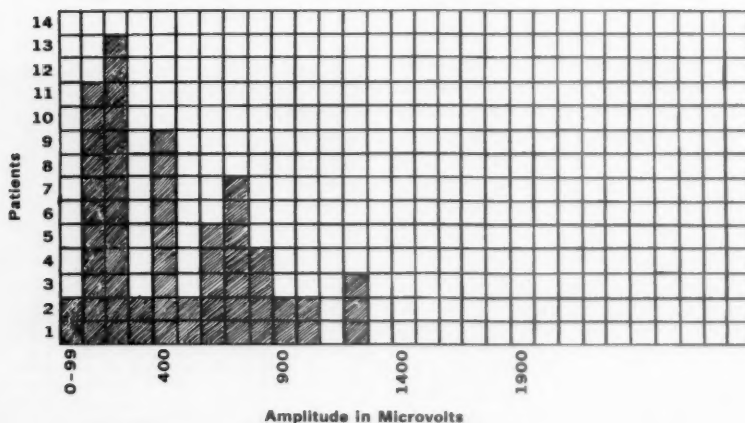


Table 2: Amplitude of Stretch Reflex — On Medication



respectively. The sharp action potentials accompanying these contractions had the same form and latency of those evoked clinically by eliciting the ankle and the knee jerks. It was concluded that the deflections in both instances were subserved by the same type of nerve fibers and expressed the same type of reflex contraction.

The significance, the variation and the characteristics of the Hoffman reflex in normals and in patients with upper motor neuron disorders has been recently reported in detail by Magladery.⁶⁻⁸ The common sequence of events recorded electromyographically from the gastrocnemius muscle on stimulation of the posterior tibial nerve in the popliteal fossa are illustrated in figure 3. With subliminal stimulation, an action potential is recorded from the gastrocnemius muscle about 30 to 32 milliseconds after the stimulus artefact. This corresponds to the reflex response of Hoffman and it is commonly referred to as the H wave. It usually will appear before any direct contraction of muscle is evident, and it is due to the stimulation of low threshold afferent fibers of high velocity. With stronger stimuli, efferent motor

fibers as well as afferent will be directly activated in the mixed posterior tibial nerve, and a second action potential will make its appearance about four to five milliseconds after the artefact. This corresponds to the direct muscle response. By progressive increase of the stimulus intensity, the reflex induced action potential reaches a maximum, then starts to decrease and finally disappears completely. At the same time, the direct response progressively increases in amplitude to reach a maximum with supramaximal stimuli. Since impulses are conducted centrally as well as distally in the motor axones, the dwindling and the disappearance of the H reflex response is attributed to the blocking effect of the antidromic impulses in the efferent axones on the reflexogenic afferent volley.

The amplitude of the H reflex, being proportional to the number of activated muscle fibers and therefore to the number of motor units that are reflexly excited, could serve as a useful and reliable indicator of spinal cord reflex excitability. If this is true, the hyper-reflexia seen on clinical examination should be accompanied by a greater amplitude of H

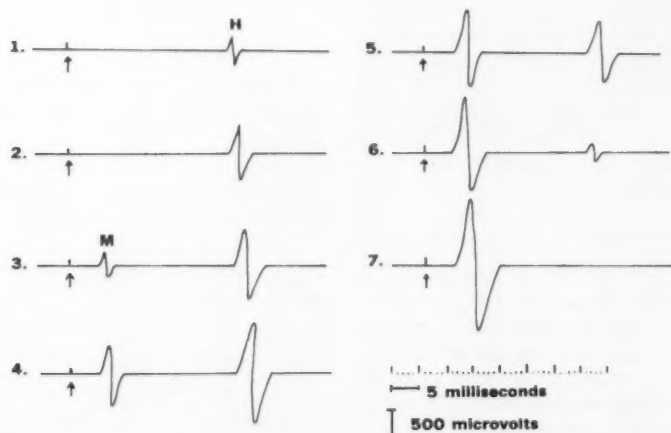


Fig. 3 — Hoffman Reflex. Characteristic sequence of amplitude variations of the reflex and direct response of the gastrocnemius following shocks of increasing intensity to the posterior popliteal nerve. The arrow points to the stimulus artefact. At subliminal impulses the reflex response (H) is obtained first after about 32 milliseconds latency and progressively increases in amplitude (1-2). With stronger intensities, due to direct stimulation of the efferent motor fibers, the direct response (M) appears after a latency of about 5 milliseconds (3-4). At supramaximal stimuli (7), the direct response is only evident. The afferent reflexogenic impulses are blocked by the antidromic volley in the efferent motor fibers. Calibration: 500 microvolts. Time: 5 milliseconds.

response. To test this hypothesis we have examined a group of 22 hemiplegic patients. The maximum amplitude of the H reflex was investigated in the spastic and normal side of the same patient so that the unaffected side served as the patient's own control.

The reflex to the calf muscle has been investigated because of the easy accessibility of the posterior tibial nerve in the popliteal fossa. The patient lies comfortably in a supine position, the knee slightly bent to about 10 degrees and with a support under the thigh. Surface recording electrodes are fixed to the skin overlying the muscle belly and equally positioned in both legs. The muscle action potentials are led to the amplifier and to a cathode ray oscilloscope. Stimulation of the mixed posterior tibial nerve is carried out through the intact skin, using two small metal disc electrodes, about 1 cm. in diameter and 3 cm. apart. The stimulus shocks were about 0.5 milliseconds in duration. The stimulating electrodes are secured longitudinally in the middle of the popliteal space and a complete recording obtained. Since the anatomical position of the posterior tibial nerve may vary, we have scanned the popliteal space by moving the stimulating electrodes to both sides of the midline and recorded the reflex in each new position. A point is always found where the nerve is maximally stimulated and where a maximal reflex response is obtained. This latter value may be considered indicative of the maximum reflex excitability of the motor neuron pool. The procedure is well tolerated despite strong current intensities that are required for supramaximal stimuli.

In tables 3 and 4 the maximum amplitudes of Hoffman reflex in both the right and left leg of hemiplegic patients are reported. The reflex response has been found to be always greater on the hemiplegic side and the difference is reported in the last column of the tables. In right hemiplegic patients the difference ranged from a minimum of 800 microvolts to a maximum of 6500 microvolts, and in left hemiplegics from a minimum of 500 microvolts to a maximum of 4800 microvolts.

Table 3: Maximum Amplitude of Hoffman Reflex in Microvolts

Patients	Right Hemiplegia		Difference
	Left Leg	Right Leg	
1	3500	8000	4500
2	5000	9500	4500
3	—	800	800
4	800	2500	1700
5	6000	8000	2000
6	5500	11500	6000
7	1650	4500	2850
8	2000	4000	2000
9	5000	11500	6500
10	2500	4500	2500

Table 4: Maximum Amplitude of Hoffman Reflex in Microvolts

Patients	Left Hemiplegia		Difference
	Left Leg	Right Leg	
1	6000	1200	4800
2	4000	1500	2500
3	3000	1500	1500
4	5000	400	4600
5	8000	6500	1500
6	10000	7500	2500
7	5500	5000	500
8	900	400	500
9	8000	6000	2000
10	8000	4500	3500
11	3500	2000	1500
12	6000	3000	3000

If the average of the various action potential amplitudes of the spastic and normal side is plotted against stimulus intensity, a curve is obtained (fig. 4) which shows the greater increase of the reflex response in the hemiplegic side.

The larger amplitude of the reflex action potential response on the spastic side is another indication of the greater number of anterior horn cells that are reflexly activated. In contrast to descriptive clinical terms this method of measurement, allows a "quantitative" determination of the exaggerated cord activity. It permits us to express spasticity numerically in terms of the difference in microvolts, between the normal and spastic side.

The usefulness of the method to assess the therapeutic effect of a muscle relaxant on spasticity has been tested.

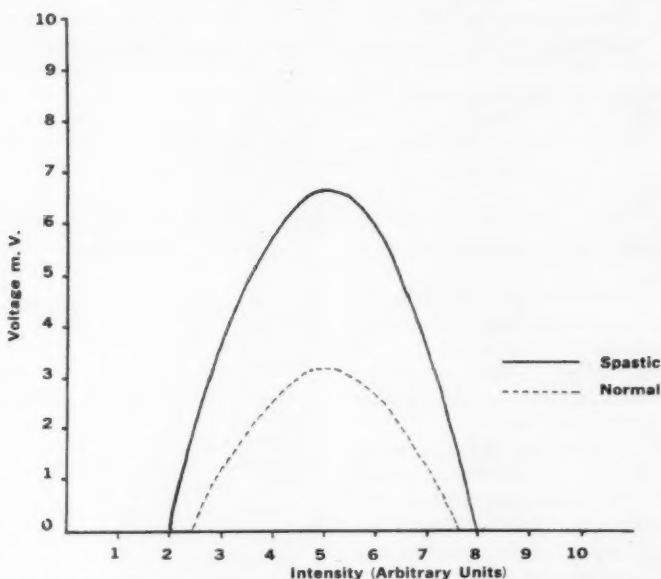


Fig. 4 — Hoffman Reflex. Curves of difference between normal and spastic muscle.

Positioning the patient and the electrodes as described above, the maximum Hoffman response was obtained. With the stimulus remaining constant, the H response persists considerably constant even for a long period of time, provided the patient is comfortable, the leg under test is properly immobilized, and the electrodes are adequately secured.

The stimulating impulses were substantially separated (one every one to two minutes) to avoid the depressing influence of closely spaced stimuli on the reflex.⁴ An intravenous spasmolytic preparation, *methocarbamol injectable*, was given to a group of patients. Ten to twenty minutes after the injection, a drop in the amplitude of the reflex response was observed in most of the cases. A full account of these results will be reported as part of another study.

The drop in amplitude was variable but always significant. A typical case is shown in figure 5, where a decrease of the reflex response amplitude of 2000 microvolts is evident after 15 minutes.

Summary

Clinical impression and observation of improvement of the patient's function

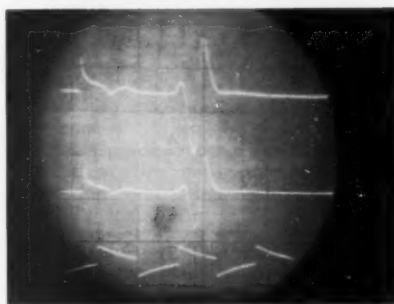


Fig. 5 — Decrease of the Hoffman reflex action potential amplitude, before (top line) and 15 minutes after (second line) intravenous administration of Methocarbamol. Calibration: 1000 microvolts. Time: 20 milliseconds.

are the criteria usually deemed sufficient to decide the value of a particular therapy in influencing spasticity. While the increase in function (real and not conjectural) is of ultimate importance in rehabilitation, such improvement may not be a direct consequence of reduction in spasticity. To avoid confusing concepts and ideas and for a better understanding of those procedures, physical or pharmacologic, commonly employed in reducing spasticity, it is necessary to have a method to test spasticity and only spasticity, as accurately and objectively as possible.

The two methods hereby presented are a step in this direction. They are based on the reflex excitability of the motor neuron cells in the cord to standardized, afferent impulses as reflected in the amplitude of the muscle action potential response. The first method makes use of a criterion generally used in clinical practice to assess spasticity; the elicitation of stretch reflex. The arrangement devised by us allows a quantitative, objective, reproduction of the phenomenon. The second method is based on the measurement of the maximal reflex response (H reflex) obtained by the electric stimulation of the afferent fiber of the posterior tibial nerve.

Clinical applicability was a major determinant in the development and application of these two technics for measuring spasticity. Too often, objective measurement technics proposed in clinical practice are impractical because the procedure is too involved or the equipment too complicated or the test places too much stress on the patient. We are not implying here that the method to assess spasticity need be as simple and reliable as the one to measure blood pressure. We do want, however, to emphasize that such laboratory procedures should fulfill some essential prerequisites of applicability in a clinical setting and not remain confined to the research laboratory. Among the essential prerequisites are the simplicity of the method, the ease of performance, the accuracy of reproduction, the ease of interpretation, and finally the minimal stress placed on the patient.

The methods we are presenting are based on well-known neurophysiologic concepts. We have applied them in the study of spasticity and have developed adequate technics and criteria so that valid, reproducible, and objective information may be obtained.

References

1. Magoun, H. W., and Rhines, R.: Spasticity: The Stretch Reflex and Extrapyramidal Systems, Springfield, Ill., Charles C Thomas, Publisher, 1948, p. 67.
2. Miglietta, O., and Lowenthal, M., to be published.
3. Mountcastle, B. V.: Reflex Activity of the Spinal Cord, in Medical Physiology, p. 1028, edited by Philip Bard, St. Louis, C. V. Mosby Company, 1956.
4. Lloyd, D. P. C.: Cited by Fulton, J. F., in Physiology of the Nervous System, p. 123, New York & London, Oxford University Press, 1951.
5. Hoffman, P.: Untersuchungen über die Eigenreflexe (Sehnenreflexe) menschlicher muskeln, Berlin, Julius Springer, 1922.
6. Magladery, J. W., and McDougal, D. B., Jr.: Electrophysiological Studies of Nerve and Reflex Activity in Normal Man. Bull. Johns Hopkins Hosp. 86:26 (May) 1950.
7. Magladery, J. W.; Teasdale, R. D.; Park, A. M., and Languth, H. W.: Electrophysiological Studies of Reflex Activity in Patients with Lesions of the Nervous System. Bull. Johns Hopkins Hosp. 91:219 (Oct.) 1952.
8. Magladery, J. W.; Porter, W. E.; Park, H. M., and Teasdale, R. D.: Electrophysiological Studies of Nerve and Reflex Activity in Normal Man. Bull. Johns Hopkins Hosp. 88:499 (June) 1951.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



Out of nothing nothing can come,
and nothing can become nothing.

— PERSIUS

Special Rehabilitation Services in Certain Cardiac and Pulmonary Disabilities

Charles D. Shields, M.D.
and

Margaret M. Kenrick, M.D.
Washington, D. C.

● During recent years improved diagnostic services along with improved surgical technics have increased the number of patients who are candidates for surgical procedures in cardiac and pulmonary disabilities. Many physicians with different types of training and numerous co-professional groups render essential services so that every patient will obtain the best results. Just as the indications for surgical procedures differ with each patient so may the rehabilitation services that are needed. All physicians concerned with the care of the patient are concerned with obtaining improved functional capacity. The physiatrist must understand the alterations in physiologic processes that result from conditions and diseases within the chest and from their surgical correction. The physiatrist must evaluate the patient prior to surgery to determine the status of his body mechanics. He also must be familiar with the social status of the patient, how this has been effected by his condition and what new hopes are stimulated by successful surgery or other medical service. Physical therapy and rehabilitation nursing should be introduced to the patient before the surgical procedure. After the operation physical therapy to improve body mechanics and to re-establish thoracic balance is indicated. Occupational therapy is essential to evaluate and to improve work tolerance and to introduce the patient to new horizons of activity. Social service and rehabilitation nursing have splendid opportunities for service to the patient after surgery. The patient who is a candidate for surgical procedures in cardiac and pulmonary disabilities will obtain best results when he receives complete rehabilitation services along with the best medical and surgical care.

Medical and surgical conditions within the thoracic vault reduce functional capacity of many individuals for varying periods of time. In recent years the thoracic cavity is entered with greater frequency and familiarity by highly trained surgeons. Tissues are removed, revised and replaced with artificial devices. Diagnostic methods, drugs, therapy and rehabilitation technics have improved. As methods have been expanded the numbers of highly trained professional groups concerned with rendering these medical services have increased. The management of intrathoracic conditions truly is an area where multiple medical services are necessary to insure the best interests of the patient and to maintain and improve medical standards.

In this presentation it is our purpose to discuss the responsibilities of physical medicine and rehabilitation. Our goal is to maintain or improve the relationship between pulmonary ventilation and the metabolic demands that are imposed upon it. We are also concerned with

1. Specific muscle weakness;
2. Joint range of motion;
3. Deformity;

4. Tightness of fascial planes;
5. Segmental alignment;
6. Functional capacity of the patient; and
7. Work capacity of the patient.

It is necessary that the patient be evaluated as early as possible and prior to surgical procedures. This establishes a standard of structure and function that may be used as a base line to aid our judgment as therapy progresses. There must be reasonable goals of accomplishment established for each individual patient. When maximum benefits are realized the patient is discharged with adequate training and instruction to properly meet his immediate needs in the area of his disability.

Physical Medicine and Rehabilitation — Prior to Surgery

Prior to a surgical procedure upon the chest, the following evaluations should be made:

Social — The patient who has an illness severe enough to be considered for chest surgery has emotional reactions that increase his burdens. An effort to determine how the illness has affected his past life and how cure or improvement will benefit his future usually is deeply appreciated by the patient. More than anything else it impresses him with the fact that the illness or operation is an episode in his life and that he may expect assistance in developing his future plans. This interview is best accomplished by a skilled social worker, closely associated with the medical evaluation group. On occasion psychiatric evaluation may be indicated.

Medical — The patient must be studied by the internist, surgeon, anesthetist and

Professor and Chairman, Department of Physical Medicine and Rehabilitation, Georgetown University Medical Center.

Assistant Professor, Department of Physical Medicine and Rehabilitation, Georgetown University Medical Center.

Read at the 3rd International Congress of Physical Medicine, Session on Medical Problems Diagnosed or Treated by Physical Medicine, Washington, D. C., August 25, 1960.

physiatrist prior to surgery. All of these physicians should review the social evaluation so that they will understand the needs and goals of the patient. The patient is given a complete history and physical examination. Of primary importance to the physiatrist's history is the type of work and play activities usual for this patient. These will assist him in evaluation of cardio-respiratory-tolerance. On physical examination the vital signs should be noted. His general appearance, mental alertness and behavior are important. One must evaluate the position of the mediastinum, the breath sounds, the rib motion and the excursion of the diaphragm, the resting position of the thorax and the symmetry of the chest. Chest expansion is best measured at the nipple line as it can be duplicated here with the greatest accuracy. The measurements should include resting position, maximum inspiration and maximum expiration. Motion capacity of all spinal and peripheral joints, tightness of fascial planes, presence of deformity, motor weakness about the spine, shoulder girdles and chest must be determined. Functional capacity if not adequately determined by the history and the physical examination may be tested in one of the therapy sections. Pulmonary function tests, which have been given before the physiatrist's examination, are noted.

The problem patients are admitted to the hospital for a short period of diagnostic study. Evaluation tests are performed. A conference is held and a plan evolved. If surgery is considered not feasible for the patient at this time but it is determined that his condition can be improved, the patient is discharged on a home program that he can follow as an out-patient. He is re-evaluated at a later date when the final decision is made. Hatch, et al,¹ in a study of 26 patients with obstructive pulmonary emphysema severe enough to make thoracotomy hazardous showed that 14 were rehabilitated by broncho-dilators, expectorants and breathing exercise so that a surgical procedure could be accomplished.

Finally complete medical and laboratory data are reviewed to determine the general condition. A plan is formulated

as to the type and extent of operative procedure, the anesthesia to be used and the type of probable postoperative needs. An understanding will be reached on how to approach the patient with firmness, caution or encouragement. The patient is routinely instructed by the physical therapist in bed positioning and in specific therapeutic exercises including breathing exercises. The reasons for all the procedures are thoroughly explained to the patient prior to surgery to insure his complete cooperation.

Breathing — The Problem

The mechanism of breathing is most complex. Metabolic activity produces its effects on the respiratory center by increase in carbon dioxide in the blood, by increase in hydrogen ion concentration or by lack of oxygen in the blood. Afferent fibers are concerned with regulation of rhythm of breathing and protective measures such as the cough reflex and protection from distention and collapse of the lungs.

Patients usually are treated with physical methods to improve the volume or depth of respiration. Mechanism of breathing is usually satisfactory and there is seldom a problem with regularity or rhythm.

Our efforts in physical medicine are designed to increase the breathing area by increasing the size of the thoracic cavity which has been reduced by disease or as a result of a surgical procedure. Our results are usually more effective with those conditions associated with surgery. This is because surgical procedures usually result in a loss of breathing area which can be readily increased by these methods. It implies that basic research in the mechanics of breathing will improve our technics.

Vital capacity, inspiratory capacity, expiratory reserve volume, residual volume, functional-residual capacity and total lung capacity are evaluated. Many arguments pro and con are made about the use of vital capacity as a method of testing and following a patient have been offered by several authors.^{2,3} In spite of its disadvantages we have found

it the most consistent method of following the patients.

With its limitations it is a simple test to perform and serves well as a base line pre-operatively, to begin therapy and to measure progress. Vital capacity is the sum of tidal supplemental and complementary air. We must keep in mind that vital capacity measurements do not indicate the amount of air present in the Alveoli and that our efforts only indirectly affect Alveolar air. Alveolar air is a physiologic and not an anatomic entity that describes air in contact with the respiratory epithelium in the alveolus which carries out gaseous interchanges with the blood. It is our purpose to use physical methods to provide more space for this function.

Surgical Aspects

Gregg⁴ and Krout⁵ discuss in recent papers how changing trends in chest

surgery for tuberculosis are less deforming than previously, making it easier for us to obtain good results. We must, however, maintain healthy respect for the surgical procedures that necessitate entering the chest cavity.

There are many surgical methods in thoracic and cardiac surgery. It is important that the physiatrist know the method and technic of the procedure involved. It is impossible to explore all approaches in chest surgery but a general outline of the anterior and posterior approach will assist in understanding the structures involved in such procedures.

In the anterior⁶ and anterolateral incision there is very little disturbance of muscle tissue. The pectoralis major is incised along the line of the skin incision (insert, fig. 1) and the major part of the muscle is retracted with the skin (fig. 1). The lower muscle bundles of the pectoralis minor are sectioned. The incision

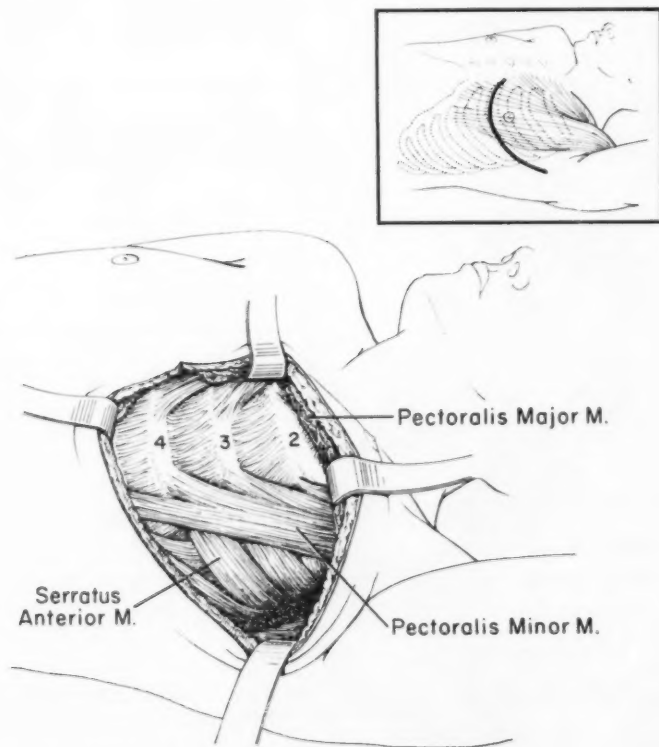


Fig. 1 — (Insert) Skin and pectoralis major incision in anterior approach. (Below insert) Anatomy after skin and pectoralis major are retracted.

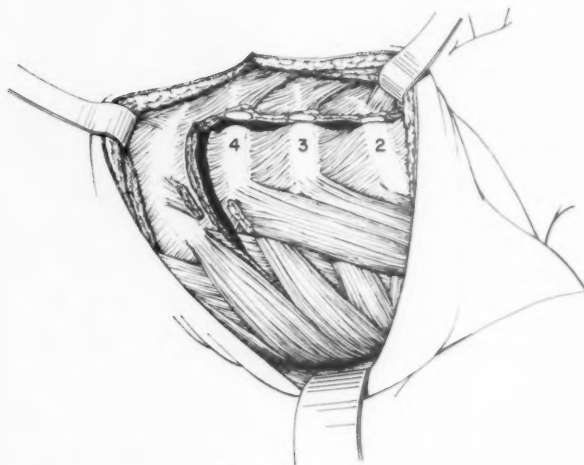


Fig. 2 — Entrance into the chest through the fourth intercostal space.

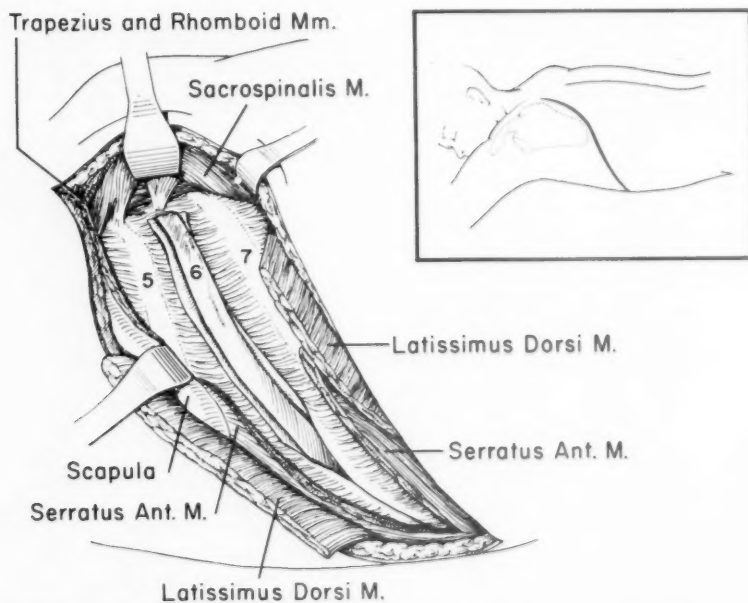


Fig. 3 — (Insert) Skin incision in posterior approach. (Below insert) Anatomy after muscles resected in preparation for rib removal.

into the plural cavity is made between two ribs. The extension posterolaterally of this incision preserves the serratus anterior by blunt dissection between the digitations of origin of this muscle. The costal cartilages are divided as is necessary for adequate exposure (fig. 2).

Closure is by suturing the costal cartilages. Pericostal sutures around the

ribs adjacent to the intercostal space which was opened return the ribs to close approximation. The pectoralis major muscle is then brought down and reapproximated separately. The superficial fascia and subcutaneous tissue are closed followed by skin.

The posterior incision begins between the spine and scapula, curves around the lower tip of the scapula and ends about the mid-clavicular line (insert, fig. 3). The lower trapezius, the rhomboid major, the latissimus dorsi and serratus anterior must all be sectioned through the muscle bellies. Usually one rib is resected. The sacrospinalis muscle is retracted so that the rib can be divided close to the transverse process (fig. 3). Small segments of ribs above and below are resected adjacent to the transverse processes as is necessary for proper exposure.

For the closure interrupted sutures are placed in the intercostal muscles adjacent to the rib which was resected. Sections of the sacrospinalis in this area are sutured to the intercostal muscles (fig. 4). The rhomboids, then the trapezius and serratus anterior are closed with interrupted sutures (fig. 5). The latissimus dorsi is then sutured. The subcutaneous tissue and skin then completes the closure.

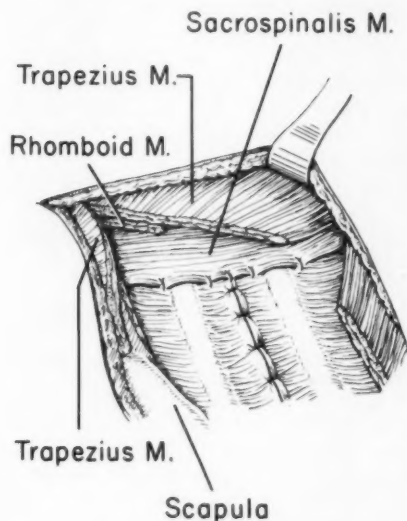


Fig. 4 — Closure of intercostals and sacrospinal muscles.

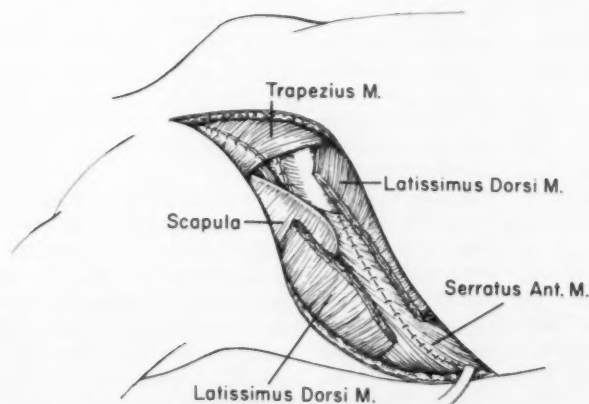


Fig. 5 — Closure of the rhomboid, trapezius and serratus anterior muscles.

Physical Medicine and Rehabilitation After Surgery

Medical Evaluation — Material in the hospital chart is reviewed with particular emphasis on events following surgery. The chest is evaluated for pattern of respiration, local areas of atelectasis or pneumonitis. Cough is evaluated for adequacy and ability to expectorate sputum. Pain is more of a problem in surgical procedures of the chest than in surgery elsewhere. This is believed to be plural in nature^{2,7} and to further hinder respiration. Breathing exercises are initiated the first day post-operatively. If done correctly these are adequate in the early stage to prevent adhesions. Other purposeful activity is not initiated until three or four days after surgery and then only after the attending internist or surgeon agrees. We prefer to start this therapy in the Physical Medicine and Rehabilitation Department because our efforts under these circumstances are more productive. On the first visit the patient is transferred by litter. In the posterior approach the long thoracic nerve may be injured. This should be checked and if a true test cannot be performed at this time it should be rechecked when indicated. Vital capacity is measured on this occasion. New orders are written for the therapists by the physician at this time. The patient receives assistance from a number of therapists but always begins with physical therapy.

Physical Therapy — The purpose of this effort is to relieve pain, begin mobilization, improve function, start ambulation and begin training in essential activities. Before any activity the therapist finds out if the patient is wearing elastic wrappings on the lower extremities. If not, these are applied with light pressure just below the knee. The purpose is to give the venous pressure a boost so as to prevent stasis until metabolic processes become more stable.

We find that the use of hot moist packs over the chest wound are valuable in relief of pain and cause more rapid healing of the wound with less conspicuous scar. These packs also are effective in initiating pressure expansion

exercises simply by means of the pressure that they exert on the chest.

Usual technics are employed to improve joint range and re-establish the scapulohumeral rhythm. Deformity usually improves with these methods along with increased expansion of the chest and relief of pain.

We use pressure expansion exercises to increase lung area. When there is marked limitation of breathing on one side of the chest, pressure is applied over the base of the lung on the affected side to establish basal expansion. With patience and effort this is often successful but if not the pressure is moved gradually higher on the same side.

On occasion, actual pressure on the sound side will aid in localization of movement on the affected side. Pressure expansion exercises have the advantage of

1. Fixing the attention of the patient on the segment;
2. Making known to him the direction and force of effort needed;
3. Acting as resistance to establish rhythm and improve strength particularly of inspiratory muscles; and
4. Mobilization of rib cage.

When expansion is established on the affected side resistance is provided in a bilateral manner to be certain that normal coordinated chest movement is present to a reasonable degree of efficiency.

The early phase of pressure expansion is done with the hands over the anterior chest. When bilateral expansion is established and the patient is sitting the posterior chest wall is included and a towel or belt may be used to supply the resistance.

These breathing technics sometimes are difficult for the patient. The patient and therapist must be relaxed and all activity preceded by careful explanation as to method and purpose. It is obvious that pressure expansion exercises have use in many surgical procedures, injuries and diseases involving the chest. The physical therapist is also concerned with mobilization of the chest cage and shoulder girdle, correction of deformity

and gradual increased activity. These activities are conducted within pain and fatigue limits.

It must be realized that the muscles of the shoulder girdle have been sectioned and reapproximated. Early range of motion exercises will prevent adhesions if coupled with minimal active assistive exercise of the cut muscles. These must be given adequate time for healing before strenuous activity is initiated otherwise the sutures will be torn and the muscle edges separated.

Occupational Therapy — As soon as the patient is capable of sitting in a wheelchair he begins a program of progressively increasing activity with appropriate rest periods designed to improve his functional ability and work capacity. We must remember that many of the patients who have undergone cardiac surgery have no idea as to the level of their new work horizon. The patient has a fixed mental level of his ability and it takes skill, training and patience on the part of the therapist to reach a new level when this is possible. This effort is preparatory to early return to work or school which is most essential for proper motivation of these patients.

Rehabilitation Nursing — The nursing section is a very essential part of the physical medicine and rehabilitation service. The nurse trained in rehabilitation techniques works with the therapists and the nursing service to insure that the patient is out of bed as long as possible each day, properly dressed, and is performing in a manner compatible with his state of recovery and training. It is her duty to see that the patient makes the most of his rehabilitation training.

It is best for this nurse to have an office in the Physical Medicine and Rehabilitation Service area where she interviews the patient and explains his obligations. She also meets with the family to discuss their responsibilities while the patient is in the hospital and when he returns to his home. The family needs instruction as to when and where the patient will need assistance and when he can function without aid. Again this exerts a stimulus to early return to work or to school.

Social Service — When the patient is performing in a fairly efficient manner the social worker confers with the patient and the physician to establish realistic and definite goals concerning work or school. She discusses problems with community agencies, employers, school and plant medical services and Departments of Vocational Rehabilitation when this is necessary.

Emphysema

Chronic bronchopulmonary disorders of the emphysema type are mentioned here because pressure type inspiration exercises are not indicated in these conditions. In emphysema the lungs are excessively distended at rest and are in a permanent respiratory position. Bronchial musculature becomes atonic in some areas of the lung and inspired air distends the bronchioles rather than enters the alveoli.

Farber and Wilson⁸ have described technics to assist the patient to breathe under these conditions. These technics utilize breathing exercises devoted to the diaphragm and chest muscles, special devices and medications and definite general instructions. Measurements of vital capacity are not of great value in emphysema to gauge improvement. In fact, it is difficult to be certain that efficiency of breathing is improved but in our experience the use of these technics help most patients to feel better and to have increased work capacity.

Summary

Patients who have surgical procedures within the chest, injuries and diseases of the chest profit from multiple services offered by the Physical Medicine and Rehabilitation Service. These activities are designed to promptly elevate functional ability, to determine work tolerance and to promptly return the patient to school, to work or to his highest level of self-care.

References

1. Hatch, Hurst B., Jr.; Bradford, J. K., and Ochsner, Alton: Value of Routine Pulmonary Function Studies in Thoracic Surgical Cases. *J. Thoracic Surg.* 34:351 (Sept.) 1957.

2. Gorlin, Richard; Knowles, J. H., and Storey, C. F.: Effects of Thoracotomy on Pulmonary Function. *J. Thoracic Surg.* **34**:242 (Aug.) 1957.
3. Miller, R. D.; Bridge, E. V., Jr.; Fowler, W. S.; Helmholtz, H. F., Jr.; Ellis, F. H., Jr., and Allen, G. T.: Pulmonary Function Before and After Pulmonary Resection in Tuberculosis Patients. *J. Thoracic Surg.* **35**:651 (May) 1958.
4. Gregg, R. A.; Sheffield, Fred J., and Mastellone, A. F.: Physical Medicine Management of Thoracic Surgery Patients. *Am. J. Phys. Med.* **36**:164 (June) 1957.
5. Krout, Robert M., and Shires, Edward B.: Physical Therapy in Thoracic Surgery. *Am. J. Phys. Med.* **34**:342 (Apr.) 1955.
6. Johnson, Julian, and Kirby, Charles K.: *Surgery of the Chest*, ed. 2, Chicago, Year Book Publishers Inc., 1958.
7. Weber, J. C.: Morphine and Amiphenazole for Postoperative Analgesia Following Major Thoracic Surgery. *J. Thoracic Surg.* **35**:105 (Jan.) 1958.
8. Farber, S. W., and Wilson, R. H.: Clinical Symposia on Pulmonary Emphysema. Summit, N. J., Ciba Pharmaceutical Products, Inc. (Nov.-Dec.), 1958.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



Men often applaud an imitation, and hiss the real thing.

—AESOP

Cancer Arthritis and Rheumatoid Arthritis

Dr. Brynjulf Strandberg

and

Dr. Niels V. Jarlov
Hellerup, Denmark

● This paper reports on 53 patients with rheumatoid arthritis, with 91 normal controls. All 53 patients were hospitalized within the last five years for the diagnosis "rheumatoid arthritis." A repeated review of the 53 patients with rheumatoid arthritis shows that they can be divided into two groups. In one group of 27 patients the Hyland's rheumatoid-arthritis test is positive in 96.3 per cent of the cases; the alkaline phosphatase is not elevated; a paper-electrophoretic study of the serum proteins shows that the 2-globulins are elevated in 37 per cent of the cases, and the titer values of antihyaluronidase, antistreptolysin, and the streptococcal antibody correspond to the values found in major series of rheumatoid arthritis patients. The other group of patients comprises 26 patients in whom the rheumatoid-arthritis test is positive in only 3.8 per cent, where the alkaline phosphatase is elevated in 80.8 per cent and the 2-globulins in 100 per cent, while the 2-globulins, antistreptolysin titer, antihyaluronidase titer and streptococcal antibody titer show values corresponding to those of the normal controls. A repeated review of the latter group of patients shows that the rheumatoid arthritis signs and symptoms can be considered as the first clinical signs of later verified cancer.

Primary progressive rheumatoid arthritis is characterized generally by the following prodromal symptoms: fatigue and general loss of strength, slight loss of weight, various nervous manifestations, slight palpitation and a feeling of muscular stiffness, particularly in the small musculature of the hand and most often in the morning. The prodromal stage develops into a more manifest stage with febrile periods, symmetrical bilateral tenderness, swelling, stiffness and restricted movement, first of the small joints of the fingers, then of the toes, foot, elbow, knee, shoulder and hip. This joint involvement, and in the above sequence, is a classic feature in a varying percentage of cases in extensive patient series of rheumatoid arthritis (lowest limit: 46 per cent, highest limit: 90 per cent). Numerous variations are found. In addition, there is muscular atrophy, tumor of the lymph glands, in a few cases tumor of the spleen, subcutaneous nodules, changes in the skin and in the connective tissue of the eye, loss of weight, loss of appetite, and the radiograph shows increasing lesions of the bones and joints. In addition, the laboratory findings change (anemia, increased erythrocyte sedimentation rate, changes in the values of the antistreptolysin, antihyaluronidase titer

and streptococcal agglutination test). Roentgenographic changes in the prodromal stage usually are not found. Swellings in the soft tissues sometimes are seen around the joints of the small fingers, but at times there are no changes. In the later stages, swelling of the periarticular tissue often is found. Still later, decalcification and osteoporosis occur, leading to destruction of the joint cartilage with narrowing of the interarticular space and increasing bone erosion, with all transitional stages to complete destruction of cartilage and bone.

The present study is an account of 53 patients who present a typical rheumatoid arthritis course, together with 91 normal controls. The 53 rheumatoid arthritis patients were hospitalized with various rheumatoid arthritis diagnoses on one or more occasions within the last five years (chronic rheumatoid arthritis, primary progressive rheumatoid arthritis, acute rheumatoid arthritis, subacute rheumatoid arthritis, etc.).

The study was inspired by Jens L. Hansen's¹ report to the Danish Physiotherapeutic Society in 1955, on the association between lung tumors and rheumatoid arthritis.

All 53 rheumatoid arthritis patients presented a clinical picture which could in all likelihood be fitted into the course of a typical rheumatoid arthritis as described above. They all showed roentgenographic changes in one or more joints; these changes also could be fitted into the picture of rheumatoid arthritis, but with the reservation that only slighter changes were observed, as is seen in the prodromal stage or in the early stages of rheumatoid arthritis.

In spite of the modest number of patients, the sex ratio in the material,

From the Department of Physical Medicine, Rheumatism and Rehabilitation, Copenhagen County Hospital.

Read at the 3rd International Congress of Physical Medicine, Session on Arthritis, Washington, D. C., August 23, 1960.

39 females and 14 males (table 1), corresponded to the percentage distribution in major collected series.

By supplementing the usual seroreactions carried out as routine in rheumatoid arthritis patients (antistreptolysin titer, antihyaluronidase titer, streptococcal

antibody titer) with Hyland's rheumatoid-arthritis-test, it was found possible to distinguish two groups of the rheumatoid arthritis patients. One group in whom the rheumatoid-arthritis-test was positive in 96.3 per cent of the cases, and another group in whom this reaction was positive in only 3.8 per cent of the cases (table 2), while the rheumatoid-arthritis-test was positive in 5.4 per cent of the control material.

Table 3 shows the variations in antistreptolysin titer, streptococcal agglutination test, and antistreptococcal-hyaluronidase titer for the two groups. It is seen that in the case of the one group of patients (27 patients), antistreptolysin titer was positive in 7.4 per cent, antistreptococcal-hyaluronidase titer in 22.2 per cent and streptococcal agglutination test in 55.6 per cent, while in the other group the corresponding reactions in the same sequence were: 11.5 per cent, 7.7 per cent and 19.2 per cent, and the values for the control

Table 1

Sex	Rheumatoid Arthritis	Cancer Arthritis	Total
Female	20	19	39
Male	7	7	14
Total	27	26	53

Table 2

Diagnosis	No. of Patients	+ Hyland RA-test	+ Hyland RA-test
Rheumatoid Arthritis	27	26 (96.3%)	1 (3.7%)
Cancer Arthritis	26	1 (3.8%)	25 (96.2%)
Normal Controls	91	5 (5.4%)	87 (94.6%)

Table 3

Diagnosis	Tests	No.	Number of + Values	+ Values in Per Cent
Rheumatoid Arthritis	AST ¹	27	2	7.4
	ASH ²	27	6	22.2
	SAT ³	27	15	55.6
Cancer Arthritis	AST	26	3	11.5
	ASH	26	2	7.7
	SAT	26	5	19.2
Normal Controls	AST	62	7	11.3
	ASH	62	7	11.3
	SAT	53	6	11.3

¹ Antistreptolysin titer > 200

² Antistreptococcal-hyaluronidase titer > 5000

³ Streptococcal agglutination test

Table 4: Electrophoresis on the Serum-Proteins

	α_2 -globulin > 9.1%	γ -globulin > 20.0%	Total
Rheumatoid Arthritis	14 (51.8%)	10 (37.0%)	27
Cancer Arthritis	26 (100.0%)	4 (15.3%)	26
Normal Controls	5 (8.3%)	8 (13.3%)	60

material were 11.3 per cent. The values in group 1 follow more closely the figures found for rheumatoid arthritis patients from other studies than do the values in group 2, which deviate significantly, especially with regard to antistreptococcal-hyaluronidase titer and streptococcal agglutination test.

In a previous study,² an estimate was made of the provisional value to be attached to the specificity of the rheumatoid-arthritis-test in the diagnosis of rheumatoid arthritis, and the diagnostic significance of positive values for antistreptolysin titer, antistreptococcal-hyaluronidase titer and streptococcal agglutination test was discussed in cases of rheumatoid arthritis. With this background, it was reasonable to assume that there was some essential difference between the two groups of rheumatoid arthritis patients. This assumption was confirmed by the results of paper electrophoretic studies of the serum proteins in these patients.

Such studies were made in all 53 patients, and the mean and normal

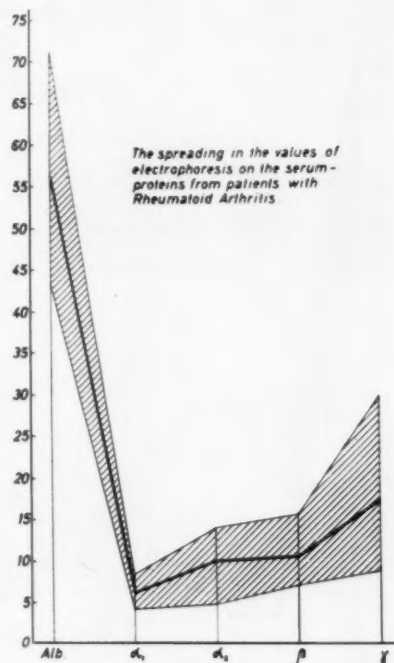


Fig. 2A

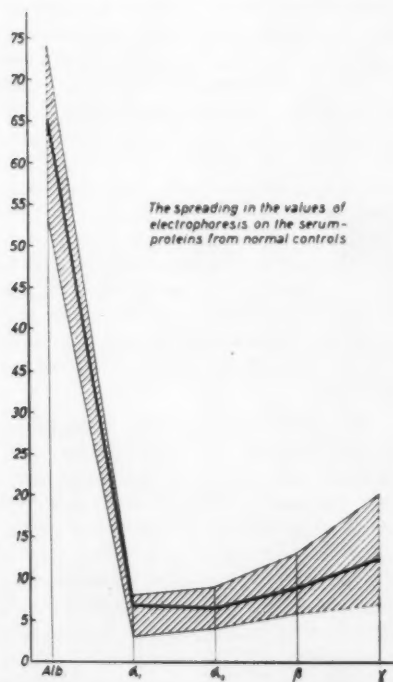


Fig. 1

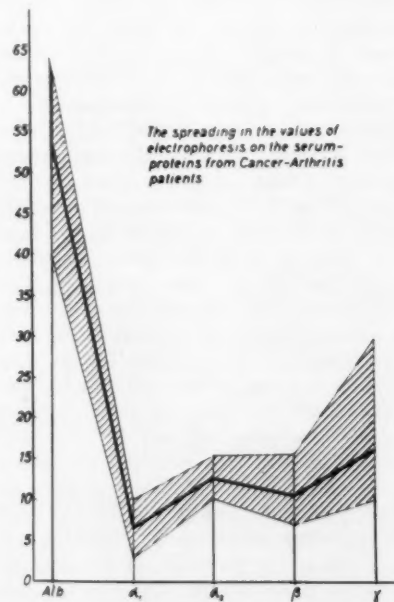


Fig. 2B

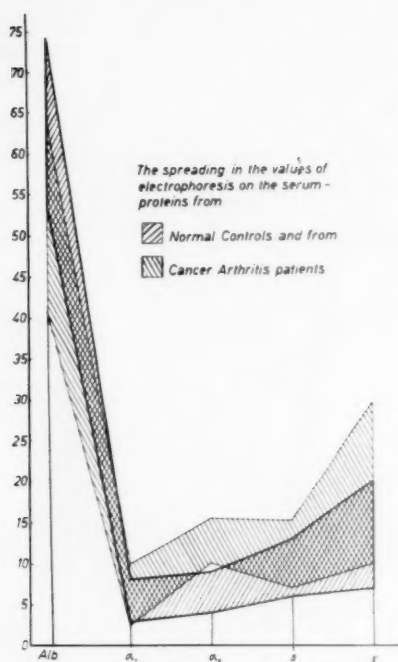


Fig. 3

maximum-minimum curve for the albumins and globulins for the control material is shown in figure 1, while figure 2, *a* and *b*, shows the same curve for the two groups of patients with rheumatoid arthritis. Figure 3 shows that the one group of rheumatoid arthritis patients (26 patients) had serum protein values which deviated significantly from those of the normal material, particularly with respect to α_2 , so that all values for α_2 in this group lay above even the highest values for α_2 in the control material. In addition, there was a considerable spread for γ -globulins and for albumins. The values for α_2 and γ -globulins, on which the curves are constructed, are given in table 4, from which it is seen that only 8.3 per cent of the control material had α_2 values over 9.1 per cent, and only 13.3 per cent had γ -globulin values which lay over 20 per cent, while the α_2 values in the one group (26 patients) of rheumatoid arthritis patients were raised in 100 per cent of the cases and in the other group (27 patients) were raised in 51.8 per cent

of the cases. In the group with 100 per cent elevation of α_2 values, the γ -globulins were elevated in 15.3 per cent of the cases, while in the group with 51.8 per cent elevated α_2 values, the γ -globulins were elevated in only 37 per cent of the cases.

In previous studies, an account was given of the value for rheumatologic diagnosis which may be attached to fluctuations in the amounts of the serum proteins as measured by paper-electrophoretic tests. It was stressed by several investigators that slightly elevated γ -globulin values and a greater spread in the values than normal are common findings but that no great diagnostic value can be attached to this, while manifest elevations in α_2 -globulins cannot be used to support the diagnosis of a rheumatic disease, but must to a greater degree result from pathologic processes of degeneration, most often from tumors. Along with the serologic differences for the two groups of rheumatoid arthritis patients, as manifest in the Hyland rheumatoid-arthritis-test, antistreptolysin titer, antistreptococcal-hyaluronidase titer and streptococcal agglutination test, the changes demonstrated in the paper-electrophoretic study of the serum proteins, particularly as far as the α_2 values are concerned, support the argument that the etiology in the one group is essentially different from that in the other, the more so as the alkaline phosphatase in the group with the elevated α_2 -globulins was elevated in 80 per cent of the cases, while it was normal in the other group of rheumatoid arthritis patients, and elevated in only 4.8 per cent of the cases in the control material.

On continued observation of the group of rheumatoid arthritis patients (26) with elevated alkaline phosphatase, negative rheumatoid-arthritis-test, and elevated α_2 -globulins, the following cancer forms were found in their initial stages: myelomatosis (1), cancer mammae (1), cancer uteri (5), reticulum-cell sarcoma (2), osteosarcoma sacri (1), osteosarcoma tibiae (1), leucaemia (2), cancer pulmonis (4), carcinomatosis pleurae (1), hypernephroma (2), cancer ven-

Table 5: Cancer Arthritis

Patient No.	Sex	ESR	Hyland RA test	Antistreptolysin Titer	Antistreptococcal-hyaluronidase Titer	Streptococcal Agglutination Test	Alkaline Phosphatases	Serum Albumina	Electrophoresis on Serum Proteins					Total Proteins	Cancer Diagnosis
									Serum Globulins						
									α_1	α_2	β	γ			
1	F	36	—	65	V/V	2000	2,2,1	10.5	48.6	4.8	70.1	7.0	29.5	5.6	Myelomatosis
2	F	23	+	80	V/V	2000	—	11.1	56.2	5.4	11.8	9.3	17.3	6.6	Cancer mammae
3	F	12	—	20	—	2000	—	14.7	54.7	5.0	10.1	13.5	16.7	6.3	Adenocarcinoma corp. uteri
4	F	22	—	65	V	1500	2,1,1	12.6	57.5	6.1	12.4	7.4	16.6	6.2	Carcinoma solidum cerv. uteri
5	F	29	—	200	V	2000	—	11.0	56.3	7.3	12.4	8.9	15.1	6.2	Reticulosarcomatosis
6	M	116	—	80	—	4000	—	25.0	50.2	7.1	13.1	11.2	18.3	7.1	Reticulosarcomatosis
7	F	13	—	20	—	2000	—	52.0	57.5	6.1	12.4	7.4	16.6	6.2	Adenocarcinoma corp. uteri
8	M	121	—	20	—	3000	2,1,1	35.0	48.5	8.7	13.2	9.2	20.4	6.0	Sarcoma ossis sacri
9	F	12	—	45	—	3000	—	35.0	59.5	7.6	13.2	9.6	10.1	6.5	Lymphatic leucemia
10	F	26	—	60	—	3000	2,1	11.5	58.8	7.2	10.8	8.9	14.3	7.0	Carcinoma pulm.
11	M	62	—	20	—	2000	—	9.7	58.9	6.2	15.2	10.7	14.7	6.7	Hypernephrom.
12	F	12	—	20	—	2000	—	32.0	52.6	6.0	11.8	8.9	20.7	6.2	Adenocarcinoma ventriculi
13	M	63	—	45	—	3000	2,1,1	13.7	39.9	8.4	14.2	13.7	23.8	6.3	Carcinoma pulm.
14	F	51	—	20	—	2000	—	17.6	47.1	9.5	13.6	13.4	16.4	5.7	Cancer corporis ventriculi (schirrosa)
15	F	67	—	120	—	3000	—	29.1	43.3	8.6	15.3	15.5	14.3	6.5	Melanosarcoma cutis
16	M	50	—	65	—	2000	—	52.0	52.0	5.5	14.0	12.8	15.7	6.8	Cancer pulm.
17	F	42	—	20	—	2000	—	11.1	59.0	5.0	13.1	10.3	12.6	6.6	Cancer colli uteri
18	F	10	—	60	—	2000	—	15.9	64.0	4.9	10.1	7.7	13.3	6.1	Carcinomatosis pleurae
19	F	39	—	60	—	2000	—	11.7	59.1	4.8	12.8	10.8	12.5	6.8	Cancer pulm.
20	M	74	—	720	—	3000	—	15.2	61.1	2.8	14.8	10.8	10.5	6.8	Cancer ventriculi
21	F	104	—	160	—	2000	—	22.2	58.3	5.9	12.4	11.7	11.8	6.8	Adenocarcinoma coli
22	F	6	—	160	—	3000	—	10.0	56.3	6.9	11.4	13.7	11.8	6.7	Adenocarcinoma ventriculi
23	F	94	—	320	—	3000	—	12.5	57.0	10.0	10.2	8.5	14.2	6.8	Hypernephrom.
24	M	11	—	60	—	2000	—	11.0	60.1	6.9	10.6	8.1	14.2	6.8	Myelogenic leucemia
25	F	98	—	60	—	2000	—	13.0	51.2	7.6	13.2	10.1	18.9	6.2	Carcinoma cerv. uteri
26	F	29	—	125	—	2000	—	17.6	54.4	6.7	12.4	10.5	16.0	6.6	Osteosarcoma tibiae
Average Values		47	—	—	—	—	—	19.3	54.5	6.6	12.5	10.4	16.0	6.5	

Table 6: Rheumatoid Arthritis

Patient No.	Sex	ESR	Hyland RA Test	Antistreptolysin Titer	Antistreptococcal-hyaluronidase Titer	Streptococcal Agglutination Test	Electrophoresis on Serum Proteins						Total Proteins	Diagnosis	
							Serum Globulins								
							Alkaline Phosphatases	Serum Albumins	α_1	α_2	β	γ			
a	F	21	+	20	—	2,1,1	2.4	60.8	6.4	9.6	10.9	12.3	5.9	Rheumatoid Arthritis	
b	F	39	+	0	—	2,2,1	3.7	62.0	6.6	7.3	7.8	16.3	7.0	Rheumatoid Arthritis	
c	F	31	+	20	—	1,1,1	2.9	56.9	6.5	11.5	10.4	14.7	5.1	Rheumatoid Arthritis	
d	M	42	+	40	—	2,2,2,1	7.1	55.8	4.7	8.3	12.0	19.2	6.8	Rheumatoid Arthritis	
e	M	46	+	100	—	2,2,2,1	8.9	50.0	8.2	8.8	10.8	22.2	7.1	Rheumatoid Arthritis	
f	M	37	+	0	—	2,2,2,1	5.8	56.5	8.3	12.6	11.3	11.3	6.2	Rheumatoid Arthritis	
g	F	36	+	50	—	0	4.2	50.8	6.4	9.6	10.9	22.3	5.9	Rheumatoid Arthritis	
h	F	19	+	0	—	2,2	7.1	61.4	6.8	8.5	10.1	13.2	6.2	Rheumatoid Arthritis	
i	F	17	+	20	—	0	2.8	65.3	5.1	9.1	8.9	11.6	5.7	Rheumatoid Arthritis	
j	F	28	+	25	—	0	5.4	61.2	6.1	9.2	11.5	12.0	5.9	Rheumatoid Arthritis	
k	F	22	+	260	—	0	5.1	47.4	8.1	14.6	11.5	18.4	4.8	Rheumatoid Arthritis	
l	F	41	+	80	—	0	6.2	47.4	8.1	12.6	11.5	20.4	5.1	Rheumatoid Arthritis	
m	F	50	+	125	—	0	7.8	62.0	5.8	8.7	9.2	14.3	7.4	Rheumatoid Arthritis	
n	F	84	+	30	—	2,1	3.5	47.8	7.6	11.2	10.4	23.0	6.6	Rheumatoid Arthritis	
o	F	31	+	110	—	0	5.7	61.8	4.5	8.6	8.8	16.3	7.2	Rheumatoid Arthritis	
p	F	31	+	20	—	1,1,1	8.8	56.9	6.5	11.5	10.4	14.7	5.1	Rheumatoid Arthritis	
q	M	19	+	65	—	0	4.6	55.7	6.9	13.7	12.1	11.6	5.8	Rheumatoid Arthritis	
r	M	9	+	40	—	2,2,2,1	2.9	61.9	7.6	9.1	9.1	12.3	6.6	Rheumatoid Arthritis	
s	F	49	+	0	—	2,2,2,1	4.3	49.8	4.6	11.4	13.9	20.3	6.8	Rheumatoid Arthritis	
t	F	73	+	30	—	2,2,1,2	5.5	48.3	5.5	9.2	9.4	27.6	6.0	Rheumatoid Arthritis	
u	F	81	+	0	—	0	5.8	54.9	5.6	8.9	9.1	21.5	6.8	Rheumatoid Arthritis	
v	F	18	+	450	—	0	5.7	55.7	4.1	6.2	9.0	25.0	6.8	Rheumatoid Arthritis	
w	M	9	+	90	—	0	4.9	71.2	4.4	7.5	8.1	8.8	6.0	Rheumatoid Arthritis	
x	F	44	+	80	—	1100	2.8	60.2	5.8	9.0	11.4	13.6	6.0	Rheumatoid Arthritis	
y	F	83	+	160	—	800	2,2,2,1	9.3	39.0	6.2	11.9	13.2	29.7	6.7	Rheumatoid Arthritis
z	M	32	+	65	—	3000	2,2,2,1	5.4	67.5	6.2	4.8	10.0	11.5	7.0	Rheumatoid Arthritis
aa	F	91	+	40	—	5000	2,2,2,1	5.3	43.3	6.8	13.9	15.6	20.4	5.5	Rheumatoid Arthritis
Average Values		40	—	—	—	—	—	5.3	56.1	6.2	9.9	10.6	17.2	6.1	

tricoli (4), cancer coli (1) and melanoma cutis (1).

At no time did any of these patients show any clinical signs or symptoms of malignant tumor, far less focal signs of some kind or extent which might suggest this. On the contrary, they showed all the clinical signs and symptoms of a primary progressive rheumatoid arthritis, for which they had in fact been hospitalized.

Tables 5 and 6 review the two patient groups.

The results of our studies endorse the opinion that, to a higher degree than previously, a rheumatoid arthritis diagnosis may be considered as a syndrome rather than a single well-defined disease. Our results also support the view that rheumatoid arthritis is a joint manifestation, which can occur as an allergic reaction to various noxious products, as in various diseases of the muscles and skin. Further, the conclusion may be drawn that the presence of rheumatoid arthritis should be a diagnostic incitement to further considerations rather than a diagnosis in itself, and finally, rheumatoid arthritis may be the first clinical sign of cancer.

Summary

The material comprises 53 patients with rheumatoid arthritis, with 91 normal controls. All 53 patients were hospitalized within the last five years for the diagnosis rheumatoid arthritis.

A repeated review of the 53 patients with rheumatoid arthritis shows that they

can be divided into two groups: one group of 27 patients in which the Hyland's rheumatoid-arthritis-test is positive in 96.3 per cent of the cases, where the alkaline phosphatase is not elevated, where a paper-electrophoretic study of the serum proteins shows that the α_2 -globulins are elevated in 37 per cent of the cases, while the antistreptococcal-hyaluronidase titer (ASH), antistreptolysin titer (AST) and streptococcal agglutination test (SAT) values correspond to the values found in major series of rheumatoid arthritis patients.

The other group of patients comprises 26 patients in whom the Hyland rheumatoid-arthritis-test is positive in only 3.8 per cent, where the alkaline phosphatase is elevated in 80.8 per cent and the α_2 -globulins in 100 per cent, while the γ -globulins, antistreptolysin titer, antistreptococcal-hyaluronidase titer and streptococcal agglutination test show values corresponding to those of the normal controls.

A repeated review of the latter group of patients shows that the rheumatoid arthritis signs and symptoms can be considered as the first clinical signs of later verified cancer.

References

1. Hansen, J. L.: Dansk Fysiurgisk Selskab. Meddelelse: 1955.
2. Eldon, K.; Jarlov, N. V., and Strandberg, B.: A New Agglutination Reaction in the Diagnosis of Rheumatic Diseases. Danish M. Bull. 6:286 (Dec.) 1959.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



Every man desires to live long;
but no man would be old.

— SWIFT

Role of Orthotics in Rehabilitation of Hands in Quadriplegia of Spinal Origin

Odon F. von Werssowetz, M.D.
Gonzales, Texas

● The essentials of any rehabilitation program for the quadriplegic patient is to provide independent function of hands with or without orthotic assistance. The selection of orthoses will depend on the severity and distribution of involvement and on the degree of complicating deformities and contractures. Most patients with quadriplegia of about the level of the sixth cervical vertebra will require adaptive orthoses. A satisfactory orthosis should attempt to replace and re-establish the normal basic hand-arm movement pattern consisting of four phases, that is reach, grip, carry and release. It is obvious that the last three are usually very severely limited. These require mechanical substitution to provide some type of holding device, and adequate stabilization of proximal joints and an independent release function. For these patients a tubular adaptive orthosis is preferred which permits easy exchange of every-day utensils which can be accomplished by the patient by gross movement without much dexterity. A new method of attachment of this holding device to the extremity was developed, utilizing hinged metal clasps on the forearm extension. These clasps are activated by leaf springs in the hinges and permit most patients to remove the orthoses independently of any outside assistance. The total result is a more functional individual.

The basic essential of any rehabilitation program for the patient suffering quadriplegia caused by injury or disease of the cervical spinal cord is to provide independent functional use of hands with or without orthotic assistance. The activities of such a quadriplegic patient are severely limited depending upon: (1) the severity and distribution of involvement and (2) the degree of complicating deformities and contractures. Some of these limitations can be modified by intelligent anticipatory or prophylactic treatment, which decreases or does not permit the chance of their occurrence.

In the early phases of this condition it is impossible to predict with any accuracy the severity and distribution of involvement. However, it is obvious that the range of functional activities will vary with the site of the lesion. Unfortunately, the roentgenographic evidence of a fracture, a dislocation or other bony lesion does not provide adequate basis for the assessment of the functional level of involvement, which may be symmetrical or asymmetrical. In addition there are often considerable deviations in the segmental innervations which may cause an apparent bizarre pattern of muscle involvement. In an analysis of muscle strength of 49 consecutive patients

with injury of the cervical spinal cord, conducted at our center by Esther Bell, O.T.R., it was found that the pattern of remaining muscular activity formed a spectrum (fig. 1) which only grossly corresponded to the usual textbook segmental levels. This spectrum listed the muscles in the sequence that they remained functional on clinical findings as resulted from standard manual muscle testing. Although there are usually no clear cut levels of involvement, for each patient there will be an area in the spectrum, above which all muscles will be strong and below which there will be little or no muscle power present.

Another factor of the greatest importance is the assessment of the remaining detector or sensory function of the upper extremity as a whole, and of the hands in particular. Here it is important to determine not only the cutaneous sensations of pain, touch and temperature, but a detailed analysis should be made of the kinesthetic and sclerotome perceptions. When these perceptions, especially the kinesthetic, are severely disturbed then the restoration of function in these patients is greatly hindered.

The residual muscle strength and the remaining perceptions are important in planning of possible goals for these patients, because these factors will help to determine the feasible functional activities, the necessity and type of orthotic supports and the success of reconstructive surgery.

Our clinical observations of quadriplegic hands revealed a significant correlation between the degree of loss of muscle power and the remaining sensory perception. It appears that when voluntary motion is absent but sensory

Medical Director, Texas Rehabilitation Center, Gonzales Warm Springs Foundation; Consultant in Physical Medicine and Rehabilitation to Surgeon General, U. S. Air Force.

Read at the 3rd International Congress of Physical Medicine, Session on Orthotics and Prosthetics, Washington, D. C., August 26, 1960.

perceptions are present, then a persistent pattern of arthropathy occurs which characteristically involves only the proximal interphalangeal joints of the fingers but not the thumb. Other joints seem to escape this complication; which consists of contractures, tightness and soft tissue swelling. As a general rule, the bony components of the joint are not involved. These changes occur in spite of persistent attempts at prophylactic mobilization. This correlation has been observed in other neuromuscular disorders, as in certain cases of hemiplegia, in which there is severe muscular involvement and only partial disturbance in perceptions. These complications do not occur when there is total absence of all perceptions.

Intelligent medical care should include among other treatment the judicious use of mobilization of hand and wrist. As a general rule the quadriplegic hand reverts to more primitive functional levels when it loses its prehensile capacity, particularly the highly developed pinch holds. This is accentuated by contractures which cause limitation of joint motion. Mobilization should maintain joints in a relatively functional range of motion but must not cause or increase instability. Often partial limitation is beneficial as it may increase the independent action of the patient. The greatest danger of developing severe contractures and tightness appears to be during the flaccid phase of the course of the disability.

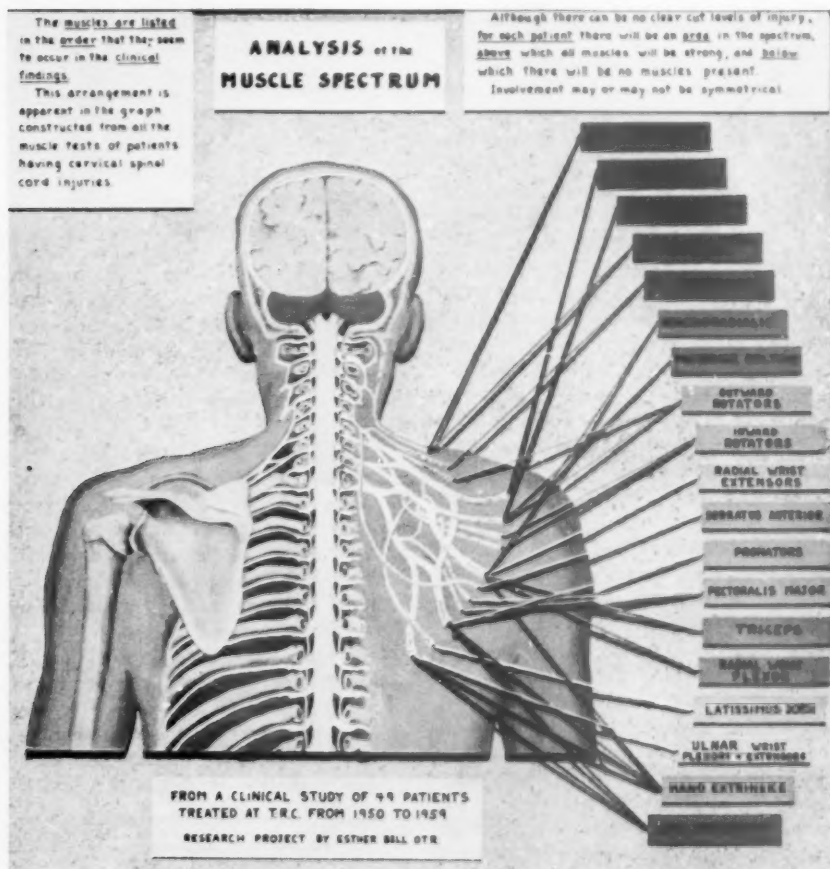


Fig. 1 — Analysis of muscle spectrum of 49 patients with cervical cord injury.

The amount of functional return in the hands also will be influenced by other complications such as spasticity, degree of reduction of vital capacity, decubitus ulcers, urinary involvement, insufficient motivation and emotional depression.

As a general rule, most of the quadriplegic patients will require some assistance to their hands, so that they can perform functional activities, because most of them have lost the normal basic hand-arm movement pattern. This pattern is composed of four phases. The first phase is the reach by which the individual is able to place the upper extremity where he desires. This phase is frequently accomplished successfully by the quadriplegic patient with spinal cord lesion at approximately the level of the fifth and sixth cervical vertebrae, because he may have satisfactory abductors and flexors of the arm and supinators of the forearm. This movement also is facilitated to some extent by gravity. However, often these patients lack pectoral muscles which interferes with their ability to perform forward motion or to reach across the front of their bodies.

The other three phases of this basic movement pattern are grip, carry, and release. Because of the muscle involvement, the performance of these actions usually is difficult, if not impossible, in most quadriplegic patients. Therefore, these deficiencies should be assessed and whenever possible corrected mechanically by adaptive orthosis. An adaptive orthosis by definition is one which either provides the hand with a holding device, or modifies certain objects and utensils so that the deficient hand can hold them adequately. In addition to this holding ability, the orthosis should provide adequate stabilization of joints but particularly of the flail wrist when present. It should have a simple mechanical release mechanism and, most of all, it should be of such a design that it will permit the quadriplegic patient, by utilizing his remaining motor power, to apply and remove it independently by himself. The orthosis should not interfere with any remaining normal or nearly normal peripheral perceptions.

There have been developed a number of different adaptive orthosis which when selected on the basis of remaining functional ability will provide help to these individuals.

If the patient has adequate control of the wrist, a fairly good motion in the metacarpophalangeal joints and some tightness in the finger flexors he may be able to use articles and utensils which are modified by two rings on top of the handles or extensions; one ring for the ring finger and one for the index finger (fig. 2). If there is some tightness in forearm rotation then the ring close to the utensil is placed under the handle for the thumb; the other ring is used for the index finger (fig. 3). Some patients will have greater functional activity with a ring-tubular attachment (fig. 4). This arrangement which consists of a hollow cylinder to which are attached one swivel ring for the index and one stationary ring for the thumb is held in the web space like a pencil. The modified handles of different utensils are slipped into this tube with ease by the patient himself.

When the hand is held in extension because of tightness of the metacarpophalangeal and interphalangeal joints of the fingers then a metacarpal cuff may help to re-establish some function. A metacarpal cuff may be of metal or leather. As a rule the metal cuffs (fig. 5) are attached to various items. The patient can slip his hand into these by himself. A modification of the metal cuff is a metal clip which is used when the fingers exhibit flexion contractures (fig. 6).

The leather metacarpal cuff (fig. 7) is made individually and consists of a leather pocket attached to a one-inch wide elastic band which keeps it in place. The leather pocket has a slot for holding utensils, most of which will fit it without alteration.

A hand, with flail digits but having relatively good passive motion in the metacarpophalangeal joints and strong wrist extensors, can be helped by a replacement orthosis (fig. 8) called by Bisgrove "the dynamic wrist extension finger flexion hand splint." The basic



Fig. 2 — Two ring modification of a "spork" (combination of a spoon and fork).

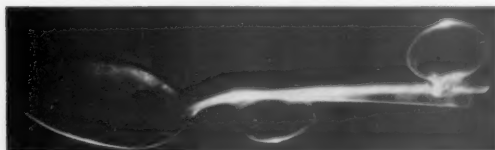


Fig. 3 — Modification of a spoon handle by one ring under and one above.



Fig. 4 — Ring tubular attachment for appropriately modified extensions or handles.

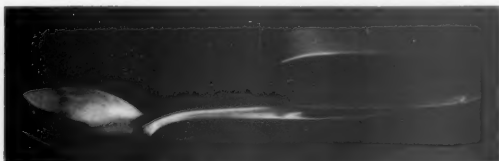


Fig. 5 — Metal metacarpal cuff.

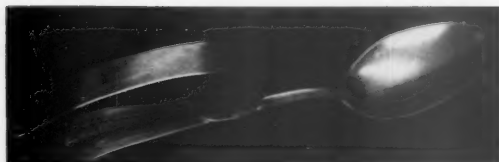


Fig. 6 — Metal metacarpal clip.

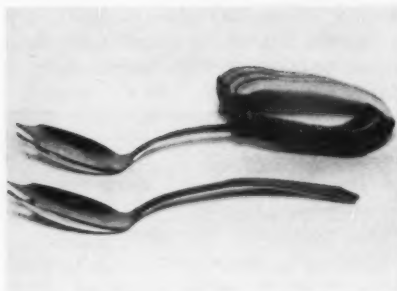


Fig. 7 — Leather metacarpal cuff.



Fig. 8 — Dynamic wrist extension finger flexion hand orthosis.

principle of this orthosis, which was described in 1916 by Souques and co-workers, is the utilization of the extension of the wrist to forcefully flex the encasted fingers against the stabilized thumb into a tripod prehensile pinch, by means of properly placed levers and hinges. The patient who is using this type of orthosis

should be advised of the danger of overworking the extensors, because when this occurs it may cause a permanent reduction in their strength with resulting impairment in function.

When the wrist is flail or shows severe involvement of the radial wrist extensor or wrist flexor then an appropriate orthosis must be used to provide stabilization of the wrist before the hand can be adapted for functional activities. Because of the low endurance and fatigability of muscles, a static support is usually indicated which would hold the wrist in a neutral or slightly dorsiflexed position. This angle is adjusted individually depending on the patient's own motion characteristics and his ability to rotate the shoulder.

For those patients who have very limited function and dexterity an orthosis was developed at our center which permits some independent function, because it provides an easy method of utilization and exchange of everyday utensils (fig. 9). The orthosis consists of a hollow metal cylinder, which is attached to a forearm extension such as a cock-up splint. This hollow cylinder, or tube, which has an inside diameter of three-eighths of an inch is placed across the palm of the hand so that it projects slightly between the thumb and index finger in the web space. Self-care articles and utensils are modified by attaching to them a metal cylindrical rod of a size that will fit the hollow cylinder of the holder. The end of this solid rod is rounded or tapered so that it fits easily into the tube. It is held securely in



Fig. 9 — Tubular holder orthosis permitting exchange of various articles.

place by a small ball tension spring lever which pushing through an opening in the tube becomes engaged in a small depression on the rod, thus causing a certain degree of friction which stabilizes the rod. The tension of the spring is so adjusted that it will permit easy removal of the utensil by exerting slight traction, such as can be made by the weight of the opposite hand or forearm. When the object is heavier then it is stabilized by a pin on the rod which is fitted to a corresponding slit on the tube.

A second shorter tube (fig. 10) is placed at an optimal angle to the first tube, between the thumb and the index finger. This arrangement permits the holding of such objects as pencils, typing sticks, etc. This tube also provides a certain degree of lateral stability to the wrist.

Clinical experience indicates that this type of holding device has a great deal of advantage over other types used,

because it is easier for the patient to slip a tapered rod into a round tube, than trying to perform this action utilizing flat holding devices.

The other outstanding feature of this tubular adaptive orthosis is the inclusion of leaf spring hinged clasps on the forearm extension (fig. 11). Most patients experience considerable difficulty in managing straps and buckles which are ordinarily used to secure hand splints in place. It also is a known fact that most patients who can wheel themselves around must remove their hand orthosis for this activity. This requires considerable dexterity and usually means some help of an attendant. This difficulty can be partly eliminated by replacing the straps and buckles by hinged metal clasps. These clasps are activated by leaf springs in the hinges which are so arranged that they will hold these clasps either fully opened or securely closed. The opening can be placed on either



Fig. 10 — Tubular arrangement for holding pencils, typing sticks, etc.

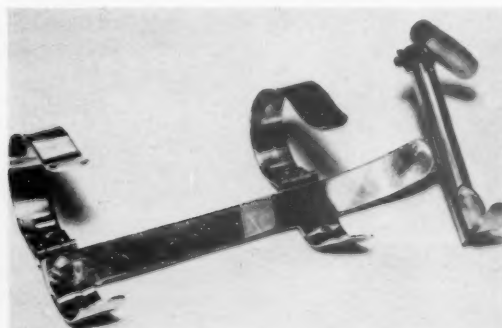


Fig. 11 — Leaf spring clasp on the forearm extension of an orthosis

the ulnar or radial side. The clasps project slightly on one end. This projection is used to open the clasp either by pushing under it or hooking it over an edge of the lapboard or the arm rest of the wheelchair. The clasp is secured by depressing it through slight pressure or weight (as of the other forearm) which trips the leaf spring in the hinge. This simple mechanism permits the patient to become independent in some hand activities because it allows him to apply and remove the orthesis by himself in most cases.

It is obvious that all adaptive ortheses have certain limitations in their practical applications. One of the major disadvantages is their limited capacity to overcome certain weight loads. When it is necessary to exert such a force then a replacement orthesis is indicated. A replacement orthesis substitutes for and replaces the activity of an entire bodily segment. It permits function by utilizing some other motor source which activates a hook or some other type of a gripping device attached to the flail hand. This motive power may be provided by some remaining functional muscles or by some external force like springs, counterbalanced weights or power derived from hydraulic, electric or pneumatic sources. To be practical this motive power must be simple to operate and must provide sufficient force for efficient prehension.

Conclusion and Summary

Orthetic supports for the quadriplegic patient must be individually selected to restore as many functional activities as

possible. The selection of the appropriate orthesis depends on the severity and distribution of involvement as determined by the muscle spectrum and remaining dermatome and scleratome perceptions, and the degree of deformities present. As a general rule, the orthesis should attempt to restore the basic hand-arm movement pattern. The patient must have an adequate source of power, muscular or external, to operate the orthesis.

The orthesis must be comfortable, have a simple design and if possible should be applied independently by the patient. The patient should understand its limitations as well as performance.

A hand orthesis which most quadriplegic patients can use satisfactorily is described. It supports the wrists and substitutes for finger grip. It has two features which permit greater independence in functional activities. It utilizes a hollow metal cylinder as the holding device into which tapered rods, attached to different items, are inserted. This permits the patient to use many items independently by being able to exchange them by gross movements. The other feature is the use of leaf spring hinges on metal clasps for straps and buckles on the forearm extension. The patient can handle these clasps independently in most cases.

In some patients it is necessary to use replacement orthesis.

Acknowledgment: The author gratefully acknowledges the technical assistance in the preparation of this paper of R. N. Witt, C.O.; Esther Bell, O.T.R.; Christine B. Stephens, O.T.R., and Rose Elliott, O.T.R., of the Texas Rehabilitation Center.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



Use of Nylon "Muscle" in Functional Bracing in Severe Quadriplegia

John S. Young, M.D.
Bruce Scott, C.O., C.P.
Virginia Gordon, O.T.R.
and
Elnora Gilfoyle, O.T.R.
Denver

• Minimum requirements for the functional use of the upper extremity are discussed. Photographic slides of a patient severely paralyzed as a result of a complete transection of the spinal cord at the level of the fourth cervical vertebra are presented showing the use of: (1) Flexor hinge splints operated by carbon dioxide activated nylon "muscles" to achieve bilateral chuck-type finger-thumb opposition. This is controlled by contralateral shoulder elevation using the trapezius muscles; (2) Ball-bearing feeders to achieve horizontal movement and rocker action; (3) Supinator aid on the ball-bearing feeder to achieve partial supination, and (4) Lastly, and the primary purpose of presenting this paper, synchronized forward flexion of the shoulder and flexion of the elbow to achieve raising one hand to the head region. This is accomplished by twin carbon dioxide activated nylon "muscles" acting upon the entire ball-bearing feeder mechanism. This is operated by a chin nudge control. A simple solution to the problem of preventing scapulohumeral rotation throughout this movement is provided.

The purpose of functional bracing for persons with severely paralyzed upper extremities is to provide controlled useful movement. Simple movement patterns which can be effectively controlled by residual innervated musculature are far more functional than a large variety of complicated movements inadequately controlled. Functional devices which require a bushel basketful of adaptive attachments and an attendant to change them do not provide satisfying nor significant independence for the patient.

This paper is essentially a case presentation illustrating basic components of upper extremity function and how they can be provided by mechanical means. The patient is a 25-year-old man who was admitted to Craig Rehabilitation Center eight years after a traumatic spinal cord injury resulting in complete motor and sensory loss below the fourth cervical cord segment. He was in excellent physical and mental condition, but completely dependent. On admission, he was able to sit in a wheelchair, but functional activities were limited to use of a mouth wand for typing. Without functional devices, he is completely dependent. A summary of his residual musculature is shown in table 1.

Basically, the upper extremity is a prehensile tool — the hand — at the end of a lever system designed to place it in position for use. The trunk is a movable foundation for this system. All three units move in a coordinated manner to produce upper extremity function.

Prehension is most simply achieved by opposition of the thumb against the finger pads of the first two fingers. The flexor hinge splint, developed by Nickel, Perry, Snelson and Staff of the Rancho Los Amigos Respiratory Center, Downey, California, provides this type prehension with movement occurring only at the metacarpophalangeal joint (fig. 1). Movement in the index and middle fingers is blocked at the distal interphalangeal joint at five degrees of flexion and at the proximal interphalangeal joint in 30

Table 1: Residual Innervated Musculature

Left	Right
Normal	Normal
0	Trace
0	Trace
Fair	Fair
0	0
Poor	Trace
0	0
0	0
0	0
0	0
0	0
0	0
0	0

Medical Director, Craig Rehabilitation Center, Clinical Instructor in Physical Medicine and Rehabilitation, University of Colorado Medical Center; Consultant in Physical Medicine and Rehabilitation, Colorado General Hospital, National Jewish Hospital, Courtesy Staff, Saint Luke's Hospital, St. Anthony's Hospital.

Consultant Orthotist and Prosthetist, Craig Rehabilitation Center.

Head of the Department of Occupational Therapy, Craig Rehabilitation Center, Occupational Therapist, Craig Rehabilitation Center.

Read at the 3rd International Congress of Physical Medicine, Session on Orthotics and Prosthetics, Washington, D. C., August 26, 1960.

degrees of flexion. Flexion occurs only at the metacarpophalangeal joint. The thumb is held firmly in opposition by a thumb post. Thus, the fingers oppose the thumb in a simple controllable movement. If no functional muscles are available in the hand or wrist, this movement can be powered by a CO₂ activated nylon "muscle" operated by any available voluntary movement. In the case of this patient, shoulder elevation is used. Opening of the hand is achieved by a rubber band extension assist.

Figure 1 also illustrates the use of this device in picking up a spoon for eating. A swivel spoon is used to compensate for absence of supination or shoulder abduc-

tion. These devices provide the patient with bilateral thumb-finger prehension. With them, he is able to pick up objects such as eating utensils, pencils, an electric razor, etc. A special drinking glass holder with a swivel mechanism to keep the glass in an upright position as it is carried to the mouth was devised. The holder for this can be seen on the thumb post in figure 1.

The wrist is held immobile by the brace in 0 to 15 degrees of extension. The hand cannot be used with the wrist extended without some element of abduction or extension at the scapulohumeral joint.

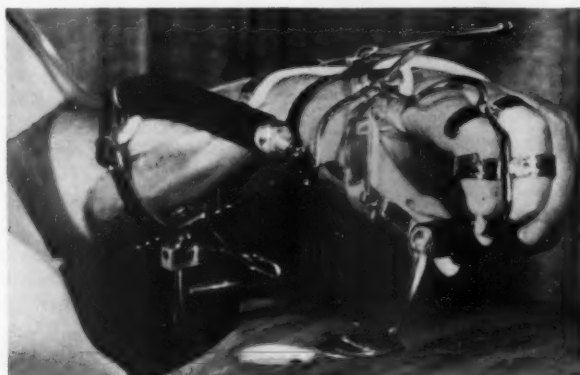


Fig. 1 — Flexor hinge splint powered by a nylon "muscle."



Fig. 2 — Range of motion in the horizontal plane. External rotation is an active motion. Internal rotation is achieved by relaxation of the muscles of external rotation against gravitational forces.

Scapulohumeral function must provide external and internal rotation and forward flexion and extension to give range of motion in the horizontal plane. Abduction is not necessary if 15 to

20 degrees of forward flexion can be provided. Compensation for severe paralysis about the scapulohumeral joint can best be achieved by the use of a ball-bearing feeder. Figure 2 illustrates the functional range of motion achieved by this patient using only a weak teres minor bilaterally. This muscle provides external rotation of the arm supported by the feeder. The movement occurs slightly uphill. Internal rotation is accomplished by gravity action controlled by the teres minor.

The rocker action of the ball-bearing feeders provides limited but well controlled movement of the hands in the vertical plane. This movement is initiated and controlled by movement of the head and shoulders.

Further movement in the vertical plane requires elbow flexion synchronized with either scapulohumeral abduction or forward flexion. Since forward flexion is also needed to move in the horizontal plane, we prefer to dispense with attempts to provide abduction. Fifteen to 20 degrees of forward flexion is necessary to supplement elbow flexion in order to achieve bringing the hand to the face region. Further, during this movement, internal and external rotation at the scapulohumeral joint must be controlled in the absence of adequate scapulohumeral stabilizing muscles.

This synchronized movement, providing for adequate range of motion in the vertical plane to allow the patient to bring the hand to the face region, is achieved by activation of the ball-bearing feeder mechanism at its proximal joint with twin nylon "muscles" (figs. 3 and 4). A rubber band assist compensates for most of the gravitational forces acting distally to the proximal joint of the feeder.

A chin nudge valve is used to activate the nylon "muscles." The valve is pushed completely down to fill the nylon tube with carbon dioxide gas. When it is pushed half way down, the gas is allowed to escape and the arm is lowered by gravitational forces. The hand can be stopped at any position within its vertical range.



Fig. 3 — Illustrating the mechanism used to produce synchronized shoulder and elbow flexion.



Fig. 4 — Patient operating mechanism to achieve movement of the hand in the vertical plane.

The top of the nylon "muscles" and the rubber bands are attached at a 45 degree angle to a steel bar which is welded at a 90 degree angle to the proximal ball-bearing feeder socket. This bar extends four inches from the bracket. The distal end of the nylon "muscles" and rubber bands are attached at a 20 degree angle to the bar of the wheelchair. As the "muscles" fill with gas, they contract approximately one-third of their length rotating the ball-bearing feeder mechanism upward. As the feeder moves upward, it flexes the elbow to 120 degrees and simultaneously moves the scapulohumeral joint through 20 degrees of forward flexion (fig. 4). To minimize friction, a double ball-bearing joint is provided between the feeder socket and the wheelchair.

As the feeder arm rises, an external rotational force is created on the proximal feeder arm. This is blocked by a metal

stop which the proximal feeder arm rests against as it rises above the horizontal plane (fig. 4). Thus, the hand controlled by the feeder mechanism is prevented from falling into internal or external rotation as it ascends in the vertical plane. Supination to almost the neutral position is provided during elbow flexion by a supinator assist on the forearm of the feeder mechanism.

An electric wheelchair, which is easily operated by the patient when the controls are placed between the wheelchair arms (fig. 3), provides the element of gross trunk mobility allowing him to place himself in position to use his upper extremities without assistance.

With very little training, the patient significantly increased his ability to function in activities of daily living.

Acknowledgment: appreciation and acknowledgment is given to the technical assistance of Donald W. Buckles.

Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.



we meet . . .

in Cleveland . . .

in 1961 . . .

hotel sheraton-cleveland

august 27 - september 1

White House Conference on Aging

Policy Statement

On Rehabilitation

Rehabilitation is the only hope for those afflicted with and disabled by chronic or degenerative conditions until such time as specific means are found to prevent and cure them. The rehabilitation program must be dynamic and total, designed to meet the physical, emotional, social and vocational needs of the chronically ill and disabled.

Only a fraction of those needing these services can secure them due to the lack of facilities, personnel, financial resources and knowledge as to the opportunities available through such dynamic rehabilitation. In an effort to meet these deficiencies and promote understanding the following recommendations are offered by the Section on Rehabilitation.

Vocational rehabilitation services must be improved and expanded so that older disabled persons may maintain or regain their ability to work and secure suitable employment. This requires staff increases; greater public understanding of career opportunities in rehabilitation; additional funds specifically allocated for the vocational rehabilitation for the aging; creation of more job opportunities for qualified older workers, continued exploration of methodology for maximum utilization of the work skills of beneficiaries receiving Social Security disability benefits; and the enactment of Federal grant-in-aid legislation to help communities establish rehabilitation workshops meeting acceptable standards to provide diagnostic and therapeutic services, and terminal employment for those not capable of competitive work. Such workshops should be eligible to secure Federal surplus property on the same basis as schools and hospitals.

Qualified teachers must be provided and well structured units concerned with the teaching of rehabilitation principles need to be established or expanded in schools of medicine, dentistry, nursing, and in departments of psychology, education, and other related disciplines, both on the graduate and undergraduate level.

Hospitals and rehabilitation centers should have adequate out-patient and in-patient facilities for the rehabilitation of the aging. Rehabilitation services could well be a condition of accreditation for hospitals particularly those with approved internship training programs.

Facilities offering long-term care should be required to have minimum rehabilitation services to be licensed or otherwise approved. Staff training programs should be available. The fee structure should be such as to encourage the provision of rehabilitation services. These facilities should be encouraged to affiliate with organized and approved training programs for the improvement of their non-professional staff. Persons with practical knowledge may serve a useful purpose to supplement the services of professional workers.

Geriatric rehabilitation must begin with the first professional exposure and persistently continue in the institution or institutions to which the patient is subsequently admitted. Prompt and comprehensive rehabilitation evaluation must be made at the earliest possible time to determine the patient's future needs.

Admission of emotionally disturbed older people to psychiatric institutions should be preceded by a thorough evaluation by qualified medical personnel.

A Federal grant-in-aid program should be established to help States develop programs designed to inculcate rehabilitation practices in governmental, non-profit and proprietary institutions providing long-term services to handicapped older people. This would involve research, demonstration and training of personnel.

Communities should develop additional supporting services and facilities such as home care programs, homemaker services, day hospitals, patient clubs, half-way houses, foster homes and preventive clinics for well older people. Careful attention to the training of the aging in the use of prosthetic and self-care devices would enable many of them to lead more independent lives.

State Councils of representatives of public agencies concerned with rehabilitation services to seriously handicapped older people should be established. Local councils should be organized also to serve as centers for public information, referral, and planning services.

Public assistance agencies should be given the financial resources to implement the provisions of their laws for self-help and self-care to include persons who are not recipients of public assistance. They should strengthen the rehabilitation concept in their medical care programs. State rehabilitation agencies should utilize existing authority to provide more rehabilitation services to older people. Arbitrary age limits where they exist should be removed. Public health agencies should be more adequately financed in order to allow them to assume a greater responsibility in the field of rehabilitation and related services.

Voluntary and other health insurance plans should provide in-patient as well as out-patient coverage for rehabilitation services in hospitals and in rehabilitation centers.

As one means of strengthening rehabilitation efforts, consideration should be given to the establishment of a National Institute of Rehabilitation.

Research should be encouraged to identify administrative and organizational patterns through which rehabilitation services can be provided effectively for older people. In this and other fields voluntary effort must be encouraged at all levels and individual community initiative emphasized. Churches, schools, libraries and other public and private institutions should be alert to the needs of older handicapped people within the scope of their own activities. There should be a Federal grant-in-aid program to help communities establish rehabilitation facilities as well as workshops.

To promote the well-being of persons suffering visual loss and maintain their integration as members of society there are available a considerable array of valid processes. These consist of two major kinds—(1) means of sight preservation and restoration, including visual aids and (2) means of self-management as blind individuals. For the first of these the combined talents of medicine, optometry and social work require special

facilities and training under the aegis of properly constituted public health programs. For the second, resources of rehabilitation are required, particularly skills in self-management as developed in rehabilitation centers for the blind, as well as the liberal use of such motivation measures as reader and guide service. Pragmatically sanctioned treatment and care as developed in various existing programs for the blind can be serviceable to the aged population, if expanded. There is need to learn more about the true function of work, leisure and recreation in the lives of aging persons with vision loss.

There is both ignorance and complacency with respect to the serious problems presented by hearing loss among the aging. There is great need, therefore, to point up the problems of the hard of hearing and the deaf and to stimulate public concern.

We need to establish a system for case finding in both urban and rural areas and to promote the training of professional and technical personnel. Hearing aids and training in their use should be provided as needed. We need also to devise special plans for job placement and job maintenance for our older people with hearing impairments.

Appropriate safety provisions for those older individuals with hearing loss should be incorporated in housing planning. Deaf persons requiring institutional care should be provided appropriate accommodations in institutions serving the aged.

Several million of our older citizens are dependent on others for meeting the normal demands of daily living. They cannot travel, feed themselves, dress, communicate adequately or move about without aid. If they were provided modern rehabilitation services, many could once again learn to live their lives in independence and with greater dignity. Some could return to work. The benefits from rehabilitation services would extend not only to these disabled persons alone, but to their families and to society as a whole. A Federal grant-in-aid program should be established to provide the rehabilitation services they need that would lead to independent living.



AMERICAN REGISTRY OF PHYSICAL THERAPISTS

ALPHABETIC LISTING

SUPPLEMENT S-1 to *Directory* (14th edition)

(From September 15, 1958 through January 31, 1961)

A

Aarestad, Darlene M., 268 Maple St., New Bedford, Mass.
 Ackerman, Toby, 135 Leyfied Terr., Springfield, Mass.
 Adams, Eldred R., Jr., 844 Main St., East Aurora, N. Y.
 Adar, Uriel, 1200 Van Nest Ave., Bronx, N. Y.
 Adler, Sylvia, 500 Sutter Ave., Brooklyn
 Agate, Marjorie A., 860 Fifth Ave., New York
 Alexander, Janna M., 6010 Greenbriar, Houston, Texas
 Allen, Agnes L., 1317 S. Dakota Ave., Sioux Falls, S. D.
 Allen, James K., Wyandotte General Hospital, Wyandotte, Mich.
 Almonney, Judy A., 140 Lookout Dr., Dayton, Ohio
 Amundson, Lee D., 631 Rockland Dr., West Palm Beach, Fla.
 Anderson, Delura Ann, 120 16th St., S., Fargo, N. D.
 Anderson, Karen J., 3522 Grand Ave., S., Minneapolis
 Anderson, Ruth P., 1231 Rose Vista Ct., St. Paul
 Anderson, Lillie G., 15 Lawn Ave., New Rochelle, N. Y.
 Anger, Rosalie E., 5200 Dupont Ave., N., Minneapolis
 Argenta, Charles N., 3213 Roosevelt Ave., Middletown, Ohio
 Ashmore, Katie C., Colonial Village, Columbia, S. C.
 Aten, Dennis W., 550 E. Platte, Colorado Springs, Colo.
 Auber, Irma L., P. O. Box 123, USAF Hospital, Wright-Patterson AFB, Ohio
 Ault, Mary M., 22 Fairlee Rd., Waban, Mass.
 Austin, Lu Anne, 615 E. Grand River, Laingsburg, Mich.
 Averill, Margaret L., 930 Cella Rd., Clayton, Mo.
 Avery, Jack L., Box 94, Cutler, Ind.

B

Bachli, Richard G., Jr., 110 Avenue "E", Ft. Madison, Iowa
 Bacon, Mary E., 1001 N. Dearborn St., Chicago
 Badell, Joab, Jr., 615 N. Tibbs, Indianapolis
 Bagley, Phyllis A., 232 W. Porphyry St., Butte, Mont.
 Bailey, Sarah Ann, 2322 Lacy Lane, Richmond, Va.
 Baker, Harrie F., P. O. Box 636, Reno, Nev.
 Baker, Julie A., 118 N. Ingalls, Ann Arbor, Mich.
 Baker, Kenneth L., 713 Iowa Ave., Iowa City
 Balke, Richard G., 1906 La Porte, Waco, Texas
 Banwell, Robert G., 2435 Flushing Rd., Flint, Mich.
 Barger, Sharon L., 1005 40th St., Moline, Ill.
 Barish, Samuel, 427 Palermo Ave., Coral Gables, Fla.
 Barner, Joyce L., 801 Joseph St., East McKeesport, Pa.
 Barnes, Robert L., 1839 Fremont Ave., Cheyenne, Wyo.
 Barney, Sharon C., 1915 Columbus Ave., Minneapolis
 Barrett, Martha L., 315 Elm St., Marietta, Ohio
 Barrows, Patricia G., 275 Superior St., S., Salem, Ore.
 Bartlett, Brooks M., 739 Beaver St., Sewickley, Pa.
 Bartlett, Helen R., 739 Beaver St., Sewickley, Pa.
 Barton, Barbara R., P. O. Box 2, Ottine, Texas
 Bates, Howard, 6631 Montague St., Philadelphia
 Bauman, Joanne F., 151 Wyndmoor, Springfield, Pa.
 Beach, Patricia E., 250 Melwood Ave., Pittsburgh
 Beam, Treva M., 1908½ 12th St., Greeley, Colo.
 Beardsley, James C., 912½ 17th St., Rock Island, Ill.
 Becraft, Joan E., 1533 W. Falkland Lane, Silver Spring, Md.
 Beger, Valerie J., 4632 Beaconsfield, Detroit
 Behrens, Claude A., Rt. 1, Box 121, Nehalem, Ore.
 Belko, Alice S., 3633a Tennessee Ave., St. Louis
 Bellak, Janet L., 401 S. Narberth Ave., Narberth, Pa.
 Belzile, Guy, 1812 S. 21st St., Terre Haute, Ind.
 Bennett, Donald E., 412 Pershing Dr., New Kensington, Pa.
 Berger, Diane H., 119 Dunmore Rd., Circleville, Ohio
 Berger, William H., 380 Dell Lane, Highland Park, Ill.
 Berglund, Barbara E., 405 E. Jefferson, Iowa City
 Berkeland, H. Bradley, 1702 S. 13th St., Fargo, N. D.
 Berriman, Dorothy R., 1757 Vista Del Valle Dr., Arcadia, Calif.
 Bethel, Martha A., 4713 Redstar, Houston, Texas
 Beyersdorf, Max A., 4531 Kansas Ave., Omaha
 Bier, Dorothy H., 2005 Wayne Ave., Dayton, Ohio
 Biesecker, Joan L., 205 Cambridge Ave., Dayton, Ohio
 Bigstaff, Mary M., 537 Aldine St., Chicago
 Billion, Charles D., Walter Reed Medical Center, 6817 Georgia Ave., N. W., Washington, D. C.

Black, Barbara A., 315 Shields Rd., Youngstown, Ohio
 Blanc, Audrey M., 1469 E. 250th St., Euclid, Ohio
 Blankenship, Jean A., 1912 Mission Ave., Carmichael, Calif.
 Blankenship, Nancy L., 519 Rue Saint Ann, New Orleans
 Blaustein, Marlene G., 314 Stubbs Dr., Trotwood, Ohio
 Blazi, Linda B., 16 E. 54th St., New York
 Blumenfeld, Diana M., 4700 Parkridge Rd., Sacramento, Calif.
 Boling, Texas H., 2808 Carol, N. E., Albuquerque, N. M.
 Bondurant, Julia K., 7645 N. Sheridan Rd., Chicago
 Bonesteel, Warren A., 7235 Penn Ave., Pittsburgh
 Borbe, Harry G., 116 Hilton Ave., Maplewood, N. J.
 Boscolo, Rhea C., Thiells Rd., Stony Point, N. Y.
 Bowlen, Dale, 551 E. Downing, Mobile, Ala.
 Bowman, Donna Lee, 436 W. 47th St., Kansas City, Mo.
 Bowman, Sarah Y., c/o Lt. Albert Bowman, 480th M. P. Det., APO 227, New York
 Boyd, Dee B., 118 S. Russell, Aurora, Ill.
 Boyer, Clifford L., 5554 N. Lakewood Ave., Chicago
 Boyer, Mary O., 3706 Wyoming, Kansas City, Mo.
 Boze, Constance R., 1605 Leighton Ave., Anniston, Ala.
 Bradley, Virginia A., 211 Loraine Ave., Cincinnati
 Bradshaw, Russell W., 5335 Belfield Ave., Philadelphia
 Brady, Glenda J., NYS Rehabilitation Hospital, PT Dept., West Haverstraw, N. Y.
 Bratton, Sherie Ann, 935 W. Colorado Blvd., Dallas, Texas
 Brayshaw, Gerald D., 10952 San Bernardino St., P. O. Box 573, Loma Linda, Calif.
 Breed, Raymond J., 1404 Maenner Dr., Omaha
 Brenner, Ann C., 929 Smith St., Buffalo
 Brewer, Joan C., Holderness School, Plymouth, N. H.
 Brice, Frederick J., 415 W. Stanford, Springfield, Mo.
 Brill, Jerome, 741 S. 60th St., Philadelphia
 Brody, Mary K., Address unknown
 Brooks, Beverly J., University Hospital, University of Washington, Seattle
 Brown, Danielle R., 826 Clermont, Denver
 Brown, Elliott, 95 Rhame Ave., East Rockaway, N. Y.
 Brown, Lois E., 4519 Pine St., Philadelphia
 Brown, Marcia A., 419 5th St., S. E., Minneapolis
 Brown, Mildred, Box 233, Letterman Army Hospital, San Francisco
 Brown, Molly L., Ireland Army Hospital, Ft. Knox, Ky.
 Buechner, Katherine M., 408 E. 18th St., Brooklyn
 Buggs, Ulysses A., Jr., 2951 South Pkwy., Chicago
 Buller, Phyllis P., Box 30, Old Zionville, Pa.
 Burgamy, William R., Jr., 763 Pine St., Macon, Ga.
 Burgette, Peggy A., 569 E. Elizabeth St., Detroit
 Burr, Alfred A., Jr., 38 Franklin Pl., Montclair, N. J.
 Russell, Dale T., Box 751, Hot Springs, S. D.
 Butler, James D., 1206 Ward St., Marlin, Texas
 Butzberger, Robert P., 1596 Cover Center Rd., Westlake, Ohio
 Buzard, Leora Ann, 1471-A Henderson Ave., Long Beach, Calif.
 Byron, Marjorie A., 151 E. 36th St., New York

C

Calabrese, Frank, 321 Pleasant View Dr., Des Moines, Iowa
 Caldwell, Karen R., 509 Hale Ave., Cincinnati
 Cammack, Joanne M., 2850 N. Cramer St., Milwaukee
 Campeau, Thomas J., 216 Jackson St., Berea, Ky.
 Canan, Betty C., 14727 Lakewood Hts. Blvd., Lakewood, Ohio
 Cannon, Virginetta S., 908 Highland Ave., Montgomery, Ala.
 Capper, Stanley L., 220 George St., Iowa City
 Cardelli, Domenic G. E., 2423 S. Hicks St., Philadelphia
 Carlson, James A., P. O. Box 274, Oshkosh, Wis.
 Carrington, Bobby T., Rt. #3, Cooper, Texas
 Carter, Nancy L., The Crippled Children's School, Jamestown, N. D.
 Casey, Rita G., 187 North St., Bristol, Conn.
 Castor, Mary E., 421 E. Walnut St., Lebanon, Pa.
 Castro, Joel A., 4311 Feagan, Houston, Texas
 Cattlett, Elsie L., RD #2, Elizabethtown, Pa.
 Chabas, Judith M., 758½ Elm St., Denver
 Chadwick, Donna B., 510 Sage, Kemmerer, Wyo.
 Chatelain, Barbara M., 1518 Barton Springs Rd., Austin, Texas
 Chepey, Frances T., 4671 Firestone, Dearborn, Mich.

Cherry, Grace E., 283 Halsted St., East Orange, N. J.
 Chevront, Laura M., 3052 W. 160th St., Cleveland
 Christian, Maria del Pilar, McKinley 104 W., Mayaguez, P. R.
 Cicero, James D., 510 W. Virginia St., McKinney, Texas
 Cichosz, Laura T., 401 E. 2nd St., Duluth, Minn.
 Clapper, Thomas D., 2506 Richwood Rd., Durham, N. C.
 Clark, Diane C., 404 N. Thayer, Ann Arbor, Mich.
 Clayton, Shelby J., 1630 Ford Pkwy., St. Paul
 Cleary, Sr. M. Therese Patricia, St. Joseph's Manor, 6448 Main St., Trumbull, Conn.
 Clemens, Leanne R., 977 Mt. Loretta, Dubuque, Iowa
 Clemenson, Shirley A., Letterman Army Hospital, Box 413, Presidio of San Francisco, Calif.
 Clement, Judith P., 848 Clarkson, Denver
 Clemons, Billy V., 1202 W. First Ave., Peoria, Ill.
 Cline, Peter D., 5954 Tchoupitoulas St., New Orleans
 Cobb, Dorothy, 250 Rockaway Ave., Boonton, N. J.
 Cohen, Gay, 1447 W. Touhy Ave., Chicago
 Cohn, Alice J., 2909 W. 7th St., Lawrence, Kans.
 Colananni, Gloria M., PT Dept., Highland View Hospital, Cleveland
 Cole, Eugene L., 103 Maple Ave., Clark Summit, Pa.
 Coles, Sheridan N., 2966 Glencoe, Denver
 Collier, H. Don, 1841 W. Masters St., Philadelphia
 Cone, James W., 1735 Foulds Rd., N., Clearwater, Fla.
 Connelly, David P., 1501 Potomac Dr., Toledo, Ohio
 Cook, George W., Jr., 900 Keith Ave., Anniston, Ala.
 Cook, Mary Beth, Box 32, Williamson, W. Va.
 Cooper, Elaine B., 15083 Steel, Detroit
 Cooper, Eleanor S., 1277 Grand Ave., San Diego, Calif.
 Cooper, Stanley B., 3700 Waggoner Ave., Evansville, Ind.
 Cornell, Elizabeth J., 326 Paloma Ave., San Rafael, Calif.
 Coryell, Jane F., 4100 Atlantic Ave., Atlantic City, N. J.
 Coulter, Jesse J., VA Hospital, Lenwood Div., Box 21, Augusta, Ga.
 Courtial, Donald C., 2550 Oakland Ave., Maplewood, Mo.
 Cowan, Hazel L., RD 1, Box 321A, Zion, Ill.
 Cravens, Marybeth W., 804 S. Jefferson, Little Rock, Ark.
 Crawford, James P., 302 Tanglewood Dr., Midland, Texas
 Crawford, Sally T., 4747 W. Alabama, Houston, Texas
 Creamer, Antonia E., 1151 E. Elm, Springfield, Mo.
 Crisci, Daniel R., 715 Matteson, Asbury Park, N. J.
 Crisp, Sylvia J., 501 Westchester, Corpus Christi, Texas
 Crissey, Ray E., Rt. 1, Box 248, Reading, Pa.
 Cronin, J. Lesley, 2421 Abrams Rd., Dallas, Texas
 Crowl, Lewis C., 2600 Johnston Rd., Columbus, Ohio
 Cunningham, Raymond E., Jr., 811 Alabama St., Bristol, Tenn.
 Curtis, Vincent C., 727 Goucher St., Johnstown, Pa.
 Custis, Sarah W., 4214 28th St., Mt. Rainier, Md.

D

Dahl, Lois M., 130 E. Woodruff, Columbus, Ohio
 Dahm, Jacquelyn F., 7567 Lindbergh Dr., Richmond Heights, Mo.
 Dahm, James P., 917 Euclid, Des Moines, Iowa
 Dalton, James C., 1509 Barrow Ave., Waco, Texas
 Daly, Herbert N., 306 W. St. Mary, Lafayette, La.
 Dammeier, Arrol Anne, Rt. 1, Box 237-e, Gig Harbor, Wash.
 Daniels, Charlotte F., 2021 Walnut St., Durham, N. C.
 Dantzer, Barbara P., 78 Haven Ave., New York
 Davis, Elizabeth P., 3736 E. 153rd St., Seattle
 Davis, Martha L., 2527 Eastgate Ave., Akron, Ohio
 Davis, Richard G., 4121 Linden Ave., Long Beach, Calif.
 Davison, Richard J., RR 2, New London, Iowa
 Davison, Robert L., 6 Kiernan Dr., Rantoul, Ill.
 Day, Barbara H., RR 1, Box 169-0, Libertyville, Ill.
 Debevec, Diana F., Box 50, 26 Adams Hill, Eveleth, Minn.
 DeChamplain, Edwina F., c/o Dr. Richard W. DeChamplain, 34th Gen. Hosp., APO 58, New York
 Dedeker, Rita M., 4222 Abner St., Los Angeles
 Deering, Elwood J., 401 Hopkins Rd., Haddonfield, N. J.
 Deibler, Phyllis C., 38 Carlisle Pl., Pueblo, Colo.
 de LaVan, Patricia L., 109 S. Harvey, Oak Park, Ill.
 Delett, Robert J., Parkway Apts., Haddonfield, N. J.
 Demkovich, Bernadine B., 4039 Chestnut St., Philadelphia
 Deml, William A., Jr., Central YMCA, Chattanooga, Tenn.
 Dennison, Genevieve R., 4071 Glenmont Pl., Columbus, Ohio
 de Rodriguez, Carmen C., 2065 Eduardo Conde Ave., Santurce, P. R.
 Derrick, Beverly, PT Clinic, Irwin Army Hospital, Ft. Riley, Kans.
 Devine, Barbara T., USAF Hospital, PT Dept., Sheppard AFB, Texas
 Devine, Richard W., 135 Pleasant Ave., Pleasantville, N. Y.
 Dicks, Diana, 5205 Tomahawk Rd., Prairie Village, Kans.
 Dillon, Herman L., West Virginia Rehabilitation Center, Institute, W. Va.
 Diment, Nancy M., 371-D Mayfair Blvd., Columbus, Ohio
 Diringer, Elizabeth C., 2221 Larkin, San Francisco
 Dobrin, Marcia, 2394 Rinard Rd., Cleveland Heights, Ohio
 Dohnanyi, Nuralon H., 341 Madison, N. E., Albuquerque, N. M.
 Dombrowsky, Ruth A., 4072 W. 56th St., Cleveland

Domy, Mardell J., 821 Fourth Ave., S. E., Waseca, Minn.
 Dontigny, Richard L., 111 E. Washington, Colorado Springs, Colo.
 Dorn, Cynthia G., 2237 N. Edward St., Decatur, Ill.
 Dorsey, Joseph A., Jr., 8 Celia St., Andover, Mass.
 Dowell, Doris J., 529 North Ave., N. E., Massillon, Ohio
 Dubiel, Norman J., Box 51, Gambier, Ohio
 Dubinski, Lawrence H., Rt. 1, S. 9th St., Walla Walla, Wash.
 Duhart, Freddie R., 1807 Lathrop St., Omaha
 Dukakis, Olympia, 39 Russell St., Arlington, Mass.
 Duncan, Edwin B., 1835 Lombardy, Houston, Texas
 Dunham, Darrol D., 1249 E. 13th St., Des Moines, Iowa
 Dupuy, Raymond J., Jr., 5550 Boaz, Dallas, Texas
 Durham, Bryan C., 344 E. 3rd N., Logan, Utah
 Durrance, James B., 1328 Warrington Way, Tampa, Fla.
 Duston, Dixie Lee, 1214 6th St., S. W., Rochester, Minn.

E

Earl, Eleanor F., 1336 Sunnymede Ave., South Bend, Ind.
 Ecker, Roland K., Hermann Hospital, Dept. PM, 1203 Ross Sterling Ave., Houston, Texas
 Edgington, Lenore, 1510 Belmar, Cleveland
 Edmonds, Richard H., 400 Dewey Ave., Buffalo
 Edwards, Eugene A., 305 W. 18th St., New York
 Edwards, Mattie I., 2603 McCall St., Dayton, Ohio
 Eisenberg, Adele C., 1119A Terrace Ave., Ft. Worth, Texas
 Eliopoulos, Charles G., 6921 Terry Lynn Lane, Cincinnati
 Ellingham, Catherine T., 652 S. 41st St., Omaha
 Ellington, Roberta S., 2860 Kincaid, Eugene, Ore.
 Ellis, Asa H., VA Hospital, McKinney, Texas
 Ellis, Nancy L., 8033 Hollywood Blvd., Hollywood, Calif.
 Ellis, Richard H., 20 Trailerville Ct., Alliance, Neb.
 Elsbree, Nancy H., 1812 S. Dupre St., New Orleans
 Elsea, Nancy K., 1803 Meadowdale Ave., N. E., Atlanta, Ga.
 Enomoto, Ruth T., 316 N. Bixel St., Los Angeles
 Epps, William H., P. O. Box 7063, Roseville Station, Newark, N. J.
 Erickson, Beverly Ann, 526 2nd St., S. W., Rochester, Minn.
 Eshleman, Donna Mae, 12332 33rd, N. E., Seattle
 Espenshade, Anita S., 2248 Augusta Rd., Greenville, S. C.
 Evans, Dianne W., 234 Lake Shore Blvd., Cleveland
 Evans, Marilyn A., 4831 Jarboe, Kansas City, Mo.
 Evans, Martha T., Box 481, Kane, Pa.
 Evans, Nancy C., 624 Orient Dr., Kansas City, Kans.
 Everett, Mary Alice, 134 White Horse Ave., Trenton, N. J.
 Exelby, Carolyn Ann, 923 Sherman Ct., Ypsilanti, Mich.

F

Faber, Mary A., 2313 W. McKinley Ave., Milwaukee
 Fair, Marguerite A., 115 Halsted St., East Orange, N. J.
 Fairchild, Ronald B., Coral Trailer Park, Coralville, Iowa
 Falor, Stanley L., 7121 Georgia Ave., N. W., Washington, D. C.
 Farrell, Catherine A., 1484 W. California Ave., St. Paul
 Federchuck, Robert A., 916 Lambeth Circle, Durham, N. C.
 Feldman, Ann, 1038 S. Main St., St. Charles, Mo.
 Fenton, Mary E., 8615 Mayfair Pl., Silver Spring, Md.
 Feyer, Marilyn A., 1315 Curtis St., Dubuque, Iowa
 Fidler, Laura P., 326 Erkenbrecker Ave., Cincinnati
 Field, Kay C., 1423 W. Langan Lane, Santa Ana, Calif.
 Filer, Barbara Lee, Crocheted Mountain Rehabilitation Center, Greenfield, N. H.
 Figley, Barbara C., Midland Hospital Assn., PT Dept., Midland, Mich.
 Fitzgibbon, Virginia L., 2123 Trevilian Way, Louisville, Ky.
 Foerster, Joan C., 1323 Ohio, Lawrence, Kans.
 Fontana, Humbert A., Jr., 3602 Edison St., Alexandria, Va.
 Fontenot, Dowell M., Athletic Dept., McNeese State College, Lake Charles, La.
 Foote, Margaret E., 415 W. 46th St. Terr., Kansas City, Mo.
 Ford, Brian C., Lucas, Kans.
 Ford, Susan G., 1008 N. W. 18th Ave., Portland, Ore.
 Fordyce, Elizabeth R., 4072 W. 56th St., Cleveland
 Foti, Joseph F., 528 5th Ave., Watervliet, N. Y.
 Fox, Gwendolyn J., 2232 Packard Rd., Ann Arbor, Mich.
 Frank, Ann J., 33 Hawthorne Terr., Leonia, N. J.
 Franks, Betty R., 75 W. Norwich, Columbus, Ohio
 Frenzel, Sr. Seraphica Marie, 3627 Vista, St. Louis
 Frey, Douglas E., 1920 Lombard St., Philadelphia
 Frickson, Lucile M., Santipara Leprosy Colony, Bongaigaon PO, Goalpara District, Assam, India
 Friedmeyer, Martha S., 3415th USAF Hospital, Lowry AFB, Colo.
 Fuchigami, Dave Y., 134-A Makawao St., Wahiawa, Oahu, Hawaii
 Fuller, Carol K., 832 Governor Nichols St., New Orleans
 Funkey, Glen G., 3216 Norfolk, Houston, Texas
 Fyffe, Beverly Ann, 560 Stanton Ave., Springfield, Ohio

G

Gage, George A., III, 1506 Chaparral Lane, Harlingen, Texas
 Galt, Lu Lynn, 483 Lucas Ct., Spartanburg, S. C.
 Gardner, Madelyn C., Address unknown
 Garman, Virginia R., 921 S. 36th St., Omaha
 Garrett, Tom R., 112 1 St. Maruale, Rochester, Minn.
 Garrity, Kathleen H., 244 E. Vernon St., Long Beach, Calif.
 Gast, Wilma Lee, 529 Palm St., Rockford, Ill.
 Gastaldo-Brac, Norma, 2703 150 St., Flushing, N. Y.
 Gault, Clay R., Address unknown
 Gay, Alda S., 1588 Ansel Rd., Cleveland
 Gay, Maryann R., 716 E. Reynolds St., Springfield, Ill.
 Getz, Linda Anne, PT Dept., The Children's Rehabilitation Instit.,
 Reisterstown, Md.
 Getzel, Rita M., 3820 Blaisdell Ave., Minneapolis
 Gibson, Joyce C., 13 Loveland St., Madison, N. J.
 Gimigliano, Arlene T., 5525 Ellsworth Ave., Pittsburgh
 Glass, Susan H., 6812 Lamar, Overland Park, Kans.
 Glick, Janet C., 919 Longford Dr., Steubenville, Ohio
 Gltzner, Alberta K., 2221 Warren St., Toledo, Ohio
 Glover, Dorothy K., Address unknown
 Goin, Patricia A., 1006 E. Walnut, Springfield, Mo.
 Goldberg, Carol J., 203 Blackhawk Rd., Highland Park, Ill.
 Golden, Walter W., 150 Goshen Ave., Elkhart, Ind.
 Goldstein, Myron W., 2020 Ave. "V", Brooklyn
 Goodman, Jack G., 1020 Third Ave., S., Fargo, N. D.
 Goodwin, Jack R., 1333 Olive, Denver
 Goodwin, John B., 2513 Ethel Ave., Waco, Texas
 Goodwin, Mary K., 2345 Post St., Jacksonville, Fla.
 Gordon, Linda G., 939 Carol Ave., Woodmere, N. Y.
 Gotthard, Ann Marie, 1588 Ansel Rd., Cleveland
 Gottdank, Marilyn B., 3287 Iowa St., Costa Mesa, Calif.
 Gould, Barbara J., 812 E. Devonshire, Phoenix, Ariz.
 Gouzd, Robert, Sr., 314 Gaston Ave., Fairmont, W. Va.
 Gracial, Lilian S., 214 E. Benbow, Covina, Calif.
 Graham, Gwendolyn M., Address unknown
 Gray, Billie Ann, 777 Meadow Sweet Dr., Corte Madera, Calif.
 Green, Irmgard, USAF Hospital, Lackland AFB, San Antonio, Texas
 Greenwald, Nancy L., PT Dept., VA Hospital, Long Beach, Calif.
 Greenwald, Neva F., 583 Harley Dr., Columbus, Ohio
 Greenwood, Robert R., 2315 Sherman Ave., Middletown, Ohio
 Gregerson, Katherine, 3427 Irving Ave., S., Minneapolis
 Grodnik, Natalie J., 445 E. 77th St., New York
 Grob, Patsy N., 118-2 Roaming Rd., Minot, N. D.
 Grobe, Joan M., 8430 123rd St., Kew Gardens, N. Y.
 Guilford, Joyce A., 12611 Riad, Detroit
 Gunter, Patricia E., Nurses Residence, Box VAH, 130 W.
 Kingsbridge Rd., Bronx, N. Y.

H

Habenicht, Charles W., 4602 Pierce St., Arlington, Calif.
 Haffner, Edward L., 98 Princeton Ave., Oxnard, Calif.
 Haines, Jeanene, 335 Peachtree Hills Ave., N. E., Atlanta, Ga.
 Haley, Ann T., 559 Howard Ave., New Haven, Conn.
 Halinski, Wanda L., 4135 E. 142nd St., Cleveland
 Hall, Gaylord L., 7346 S. E. Totman St., Portland, Ore.
 Hall, Linda Marie, 1450 Berkshire Rd., Columbus, Ohio
 Hall, Mary S., USA Hospital, PT Section, Ft. Carson, Colo.
 Hall, Nancy Lou, Box 1013, Fremont, Texas
 Hamilton, Doris W., 2718 2nd St., S. E., Washington, D. C.
 Hammer, Elizabeth A., 3403 18th Ave., S., Minneapolis
 Hammond, Charles W., Potter, Neb.
 Hanes, Martha J., 2412 S. W. 12th St., Miami, Fla.
 Hannah, Patricia F., 215 Woolf Ave., Iowa City, Iowa
 Harbine, Patrick E., 603 Plymouth, Missoula, Mont.
 Hardy, Philip O., 11247 San Juan, Loma Linda, Calif.
 Harmon, Katherine Ann, 530 N. Union, St. Louis
 Harnar, Julie, Watkins Memorial Hospital, Lawrence, Kans.
 Harris, Barbara Jean, Rt. 3, Cleburne, Texas
 Harris, Mary Anne, 8221 Shelbyville Rd., Louisville, Ky.
 Harrison, Donna J., 100 1/2 Veterans Village, Ft. Collins, Colo.
 Harrison, Richard R., 4823 Glenwood Rd., Riverside, Calif.
 Hartenbach, Suzanne E., 535 N. Mosley Rd., Creve Coeur, Mo.
 Harvill, Howard C., 1608 31st St., Galveston, Texas
 Haskew, Norma Mae, 4800 Madison Ct., N. E., Albuquerque, N. M.
 Hass, Shirley Ann, RR 3, Sac City, Iowa
 Hassan, Rita O., 3500 14th St., N. W., Washington, D. C.
 Hauser, Constance A., 60 Green Bay Rd., Winnetka, Ill.
 Hauser, Marjorie S., 500 W. 58th St., New York
 Hawkes, Ann Marie, 203 W. Valerio St., Santa Barbara, Calif.
 Haws, Claud F., Jr., 2721 S. 28 St., Paducah, Ky.
 Hayes, Susan M., 3441 W. 159th St., Cleveland
 Hayman, Johnny W., 1056 E. Falmouth St., Warrenton, Va.
 Hazel, Sylvia L., 7337 Ravellhill Lane, Dallas, Texas
 Hazelton, Frank T., 5883 Council St., N. E., Cedar Rapids, Iowa

Heath, Barbara W., 894 Riverside Dr., New York
 Heidinger, Patricia K., 1833 Roxford Rd., East Cleveland, Ohio
 Hein, Susan L., 3 Brookline Lane, Dearborn, Mich.
 Heine, Alvina L., 154 Bickley Rd., Glenridge, Pa.
 Heiser, James L., 843 1/2 Almaden, Eugene, Ore.
 Hellewell, Robert R., 1659 Kent, Ravenna Rd., Box A 24, Kent, Ohio
 Helms, Mary D., 1723 N. 11th St., Terre Haute, Ind.
 Hemenway, Virginia R., 175 Elm St., West Haven, Conn.
 Hense, Thomas E., 2200 Pillsbury Ave., S., Minneapolis
 Hessler, Donna Jane, Box 2557, USAF Hospital, Andrews AFB,
 Washington, D. C.
 Higgins, James J., 1931 Portland Ave., S., Minneapolis
 Hightower, Ann B., 303 Crescent Rd., Beckley, W. Va.
 Hill, Joanna C., 44 Gordon Rd., Milton, Mass.
 Hirsch, Judith, Goodland, Kans.
 Hirsch, Miriam K., 4243 Okalona Rd., South Euclid, Ohio
 Hochberg, Naomi J., 4 Garrison St., Chestnut Hill, Mass.
 Holt, Elaine S., Rt. 1, Marion, Ohio
 Holzum, Sr. Gerard Marie, 1536 Papin St., St. Louis
 Hopkins, Jence E., 5702 Ridgedale Dr., Houston, Texas
 Hoppens, Neil S., 1702 W. Third, Perry, Iowa
 Hosmer, Norma E., 709 S. Chapel, Louisville, Ohio
 House, Alfred M. D., Jr., Address unknown
 Houser, Randal D., 521 Jameswood Dr., Paducah, Ky.
 Houston, Barbara R., 727 Sheffield Ave., N. E., Massillon, Ohio
 Howalt, Georgene M., E. Orleans St., Stillwater, Minn.
 Howard, Thomas L., 207 W. 14th, Hutchinson, Kans.
 Howe, Sarah S., 322 N. State St., Ann Arbor, Mich.
 Hubbard, Beverly Ann, PT Section, Beach Pavilion BAH,
 Ft. Sam Houston, Texas
 Hubbard, Patricia T., 4517 Patterson, Richmond, Va.
 Hubbs, Marvin E., 1551 Willowside Rd., Santa Rosa, Calif.
 Huber, Carol Lee, 1639 Hampton Rd., Akron, Ohio
 Huey, Lois M., 200 N. State, Ann Arbor, Mich.
 Huggett, Pat G., Address unknown
 Hummer, Willa G., 1678 1/2 Lake Rd., Conneaut, Ohio
 Humphrey, L. Dennis, 1336 S. 6th St., Fargo, N. D.
 Hupprich, Emma M., 11 Lakeshore Ct., Madison, Wis.
 Hyma, Beata M., 1301 Forest, Ann Arbor, Mich.

I

Idzkowsky, Betty G., 1537 Denniston Ave., Pittsburgh
 Inman, Douglas A., 5710 35th, N. E., Seattle
 Irvin, Richard F., 910 Normal, Normal, Ill.
 Isaacs, LaVonne E., 2255 Olive, Denver
 Isaacson, Gabriella K., 2285 Broadway, San Francisco
 Izzo, Pasquale J., Richard Pl., East Norwich, L. I., N. Y.

J

Jackson, Bonnie W., 32D Brookline Manor Apts., Reading, Pa.
 Jackson, Cynthia E., PT Clinic, Walter Reed Hospital,
 Washington, D. C.
 Jacobson, Janice C., 528 W. Wellington St., Chicago
 Jacoby, Phyllis N., 7308 Kensington, Maplewood, Mo.
 Jaeger, Robert C., 905 40th St. Ct., Moline, Ill.
 Jaekle, Robert C., 21 Colony St., Seymour, Conn.
 James, Carole Ann, 378 Glenwood Ave., Akron, Ohio
 James, Doris D., 136 1/2 North Ave., Talmadge, Ohio
 James, Martha Jean, Box 744, Luling, Texas
 Janos, Mary Ann, 15776 Euclid Ave., East Cleveland, Ohio
 Jardine, Shirley Ann, 35986 Jefferson, Richmond, Mich.
 Jarrell, Betty J., 9615 Manor Rd., Kansas City, Mo.
 Jeffrey, Jacqueline A., 3610 Rainbow Blvd., Kansas City, Kans.
 Jenks, Barbara M., 4039 Spruce St., Philadelphia
 Jensen, Ione R., 2920 Panama St., Bakersfield, Calif.
 Johanni, Barbara Ann, 4209 W. 23rd St., Cleveland
 Johnson, Fritz E., 10438 Alderbrook Pl., Seattle
 Johnson, Ida J., 4611 Kemerly, St. Louis
 Johnson, Mary Ellen, 1913 Columbus Ave., Minneapolis
 Johnson, Molly J., 1551 Lexington Ct., Kansas City, Mo.
 Johnson, Odell, Jr., Box 304, 31 Cherokee, State College, Ark.
 Johnson, Robert D., 701 N. 12th St., Virginia, Minn.
 Johnston, Betty B., 11 Speradakis Terr., Cambridge, Mass.
 Jolly, Mary E., 37 Newell Dr., Bloomfield, N. J.
 Jones, Dian C., 38 West Ct., Appleton, Wis.
 Jones, Donald G., 132 Mt. Vernon Rd., Newark, Ohio
 Jordan, Eileen E., 1301 Kansas Ave., McKeesport, Pa.
 Juday, Marcia A., PT Dept., John Sealey Hosp., Univ. of Texas
 Medical Branch, Galveston, Texas
 Julian, John A., 3102 E. Osborn Rd., Phoenix, Ariz.
 Julian, Nancy R., 3102 E. Osborn Rd., Phoenix, Ariz.

K

- Kaczorowski, Janice A., 1134 17th St., Santa Monica, Calif.
 Kagler, Gail W., 504 Clinton Springs Ave., Cincinnati
 Kalisiak, La Rae, 100 Crockett St., Seattle
 Kamenshek, Dorothy M., 3695 Plainfield, N. E., Grand Rapids, Mich.
 Kaplan, Frances B., 2727 Ocean Pkwy., Brooklyn
 Karnuth, Harold A., 5000 Wilcox, Downers Grove, Ill.
 Kassell, Joann, 28 Glencoe Rd., Columbus, Ohio
 Kasten, Walter R., 622 W. Austin, Flint, Mich.
 Kauders, Diane E., 1020 Clinton St., Philadelphia
 Kazmierczak, Carol B., 2654 Scio Church Rd., Ann Arbor, Mich.
 Keen, Ruth Ann, 5512 Walnut St., Pittsburgh
 Kefover, Mary Rose, 310 Lydia St., Carnegie, Pa.
 Keisel, Glenn L., RD 1, Pettis Rd., Meadville, Pa.
 Keller, Joan, 1510 Belmar Rd., Cleveland
 Keller, Phyllis J., 612 W. Story St., Bozeman, Mont.
 Kelly, Elaine G., Box 256, Tire Hill, Pa.
 Kelsen, Ruth R., 1311 E. Grand Ave., Des Moines, Iowa
 Kempenich, Sr. M. Veronica, St. Gabriel's Hospital, Little Falls, Minn.
 Kendall, Denise D., 4005 Eaton, Kansas City, Kans.
 Kennedy, G. June, 306 W. Ninth Ave., Columbus, Ohio
 Keogh, Roderick B., 10003 Gravelly Lake Dr., Tacoma, Wash.
 Kerchner, Julie C., 40 Willett St., Albany, N. Y.
 Kerr, Donald J., 1151 Highland Ave., Ambridge, Pa.
 Kerrigan, Mary E., 952½ Kennebec St., Pittsburgh
 Kimball, Robert R., 905 Moore Ave., Lufkin, Texas
 Kimble, Lila M., 38 Estel Rd., Fairfax, Va.
 Kimble, Sheila M., 934 Edgewood Ave., Lancaster, Pa.
 King, Patricia, 4816 N. 36th St., Tacoma, Wash.
 Kirkegaard, Paul F., 1716 Pierce St., Sioux City, Iowa
 Klatt, Phyllis R., Address unknown
 Klenen, Jo Anne, 1908 Pine St., Philadelphia
 Kleszyk, Kathleen A., 124 9th St., N. E., Rochester, Minn.
 Klibanski, Mordecai, 6931 N. Greenview Ave., Chicago
 Klotz, Lois E., 1405 W. Ash St., Columbia, Mo.
 Knapton, Richard G., 226 Wenonah Pl., West Palm Beach, Fla.
 Knoerzer, Nannine L., 69 E. Bellevue Ave., Chicago
 Knox, Walter L., 1630 W. Orangeburg Ave., Modesto, Calif.
 Koch, June A., 6553 Calhoun, Dearborn, Mich.
 Koellner, Mary Ann, 1662 E. Swan Circle, Brentwood, Mo.
 Kovalchik, Barbara S., 381 E. Main St., Somerville, N. J.
 Kow, Kathleen C., 1745 S. Magnolia Ave., Los Angeles
 Kreider, Russell R., 2206 Holly St., Richmond, Va.
 Kressley, Nevin W., 809th Med. Gp., 10 Second Dr., MacDill AFB, Fla.
 Krum, Priscilla B., 2958 Cleveland Ave., Niagara Falls, N. Y.
 Kubes, Benjamin F., 507 6th St., N. W., Austin, Minn.
 Kuehl, Judith G., 475 E. Lockwood, Webster Groves, Mo.
 Kuivinen, Barbara C., 428 Oakland Ave., Oakland, Calif.
 Kulick, Jeanne F., 1494 N. High St., Columbus, Ohio
 Kurerth, Julie M., 7124 Knox Ave., S., Minneapolis
 Kurashe, Milnes C., 190 Willoughby St., Brooklyn
 Kurras, Richard A., 10421 S. W. 50th Terr., Miami, Fla.
 Kushner, Francis J., 614 Highland Ave., Chester, Pa.

L

- Ladion, Herminia J. G., Manila Sanit. & Hospital, Box 1592, Manila, Philippines
 LaDuke, Mary J., 287 N. Smith Ave., St. Paul
 Laffaw, Sally A., 607 W. 76th, Seattle
 Laird, Henry L., Box 414, Coleman, Texas
 Lalim, Andrew, 9915 Fremont Ave., S., Minneapolis
 Lamar, Patricia Ann, 146 Shawnee Pl., Lexington, Ky.
 Lane, Lauren E., 182 Saxer Ave., Springfield, Pa.
 Lane, Laurie Ann, 1940 7th Ave., Oakland, Calif.
 Lang, Robert D., 25392 Cole St., Loma Linda, Calif.
 Lang, William F., High Bank Estates, Rt. 5, Salisbury, Md.
 Langley, Diana N., 432 Hillside Dr., Big Spring, Texas
 Larkin, Lynn H., 116 Coffeen, Sheridan, Wyo.
 Larsen, Mary M., 412 W. Du Bois Ave., Du Bois, Pa.
 Larsen, Suzanne Z., 106 Bellevue Apts., Sioux City, Iowa
 Larson, Alexander U., Box 32, Sunmount VA Hospital, Sunmount, N. Y.
 Larson, Richard D., 410 E. South St., Viroqua, Wis.
 Larson, Robert L., 709 Ave. "R", Lubbock, Texas
 Laskin, Herbert, 6034 Baltimore Ave., Philadelphia
 Laubach, Barbara M., 1835 N. 39th St., Milwaukee
 Laulainen, Jacquelyn A., 1360 Monroe, Denver
 Lee, Betty H., 311 Carson St., Brush, Colo.
 Lee, Cassie L., 3426-D Southern Oaks Blvd., Dallas, Texas
 Lee, Marjorie U., 3717 Tiffany Dr., Dayton, Ohio
 Leffer, Thomas F., Jr., 7259 Glenmore Dr., Lambertville, Mich.
 Lensch, Roberta M., 830 5th St., N. W., Rochester, Minn.

- Leonard, Dorit G., 4627 W. Pico Blvd., Los Angeles
 Levine, Roberta H., 13 B Pardon Rd., North Brunswick, N. J.
 Levitan, Judith, 523 W. 187th St., New York
 Lewis, Donald W., 1225 Montana, Wenatchee, Wash.
 Lewis, Mary Ann, 100 Jackson, Denver
 Libby, Pauline M., 428 Chelsea Rd., Fairless Hills, Pa.
 Lied, Jo Ellen, 614 Woodard Dr., Kirkwood, Mo.
 Likar, Edmund P., Box 51, Meadow Lands, Pa.
 Link, Judith R., 15830 Joy Rd., Detroit
 Loar, Paul J., PT Dept., Shadyside Hospital, Center Ave., Pittsburgh
 Locker, Georgia R., 612 Church St., Ann Arbor, Mich.
 Loehr, Florence E., 1834 Brightwood, East Cleveland, Ohio
 Lofgren, Sylvia L., 1711 Pleasant St., Des Moines, Iowa
 Long, Roger W., 1439 Arthur, Des Moines, Iowa
 Lubin, Lester, 155-30 86th St., Howard Beach, N. Y.
 Lucas, Mary Elizabeth, 2110 Dexter Ave., Silver Spring, Md.
 Lupi, Frances A., 906 Belmont Ave., Utica, N. Y.
 Lynch, Gale M., 830 Van Nuys St., San Diego, Calif.
 Lystad, Anita F., 140242 Central Ave., Evanston, Ill.
 Lytle, Judith F., 17316 Invermere Ave., Cleveland

M

- MacIntosh, Martha W., c/o Capt. R. B. MacIntosh, 30th Med. Det., APO 108, New York
 Maeder, Bernice Z., 20 Hollis St., Holliston, Mass.
 Majors, Nelda F., 621 E. 27th St., Houston, Texas
 Malseed, Patricia E., NYS Rehabilitation Hospital, West Haverstraw, N. Y.
 Manders, Sandra Jean, 2001 Adolphus St., St. Paul
 Mandile, Leo S., 4213 N. Ashland Ave., Chicago
 Maniscalco, Leonard, 651 Larchmont Dr., Daly City, Calif.
 Marcus, Judith I., 18 E. 21st St., Brooklyn
 Martin, Glover, 1375 S. Salt Pond, Marshall, Mo.
 Martini, Eileen D., 8309 Burns Ave., Cincinnati
 Masato, June J., 3027 12th Ave., Los Angeles
 Mason, Paul B., Address unknown
 Matrozza, Frank S., 75 Central Ave., Turtle Creek, Pa.
 Matson, Virginia D., c/o Robert Matson, 917 N. Union Rd., Dayton, Ohio
 Mattox, Deane L., 777 Ash, Denver
 Maune, Sylvia D., 1400 Porter St., Columbia, Mo.
 Mazzacane, Richard S., Montebello State Hospital, 2201 Argonne Dr., Baltimore
 McBroom, Gretchen G., 1450 Broadview, Columbus, Ohio
 McCallum, Ann M., 311 S. Prairie Ave., Champaign, Ill.
 McCarthy, John L., 4033 Dean Dr., Oak Lawn, Ill.
 McClatchie, James G., 2918 Underwood St., Lafayette, Ind.
 McCracken, Harold L., 708 N. Negley Ave., Pittsburgh
 McDaniel, June H., 330 Texas, S. E., Albuquerque, N. M.
 McDonald, Marianne E., 3300 Diell Ave., Richmond, Va.
 McDougald, Audrey J., 2047 Jean St., Houston, Texas
 McGee, Patricia P., 2223 "H" St., N. W., Washington, D. C.
 McGinnis, Mary C., 906 S. Vine St., Urbana, Ill.
 McIlvried, Sally H., 7428 Boyer St., Philadelphia
 McInnis, Judy A., 3349 5th St., Port Arthur, Texas
 McKenzie, Gertrude O., Peerless Dr., Cleveland, Tenn.
 McKinlay, Florence D., 2500 Oriole Trail, Long Beach, Michigan City, Ind.
 McMichael, Gene L., Box 3943, CAFB, Columbus, Miss.
 McMillen, Richard C., 1815 7th St., Beaver Falls, Pa.
 McPherson, Dorn W., 3528 Moon Dr., Mesquite, Texas
 Medley, Carol Joan, 9730 Belinder, Kansas City, Mo.
 Mehus, Sandra F., 11A Scenic Dr., Orinda, Calif.
 Melder, Frankie R., 1010 W. 19th, Austin, Texas
 Mendelson, Janet G., 3184 Shady Ave., Pittsburgh
 Mercer, George K., 3703 Worth St., Dallas, Texas
 Meyer, Eva R., 4812 Nakoma Dr., Okemos, Mich.
 Meyers, Royce L., West Texas Rehabilitation Center, 4601 Hartford St., Abilene, Texas
 Mikush, Donald C., 6502 Windsor Mill Rd., Baltimore
 Miller, Jacquelyn K., 615 Walnut St., Leavenworth, Kans.
 Miller, Mervin Ann, PT Dept., James Walker Memorial Hospital, Wilmington, N. C.
 Miller, Michael, Box 2000, Porterville, Calif.
 Millow, Annamae, 1444 Church Ave., Scranton, Pa.
 Mills, Sally V., 326 Ninth, Box 70, Seattle
 Millsop, Carol Ann, 1909 5th Ave., N., Minneapolis
 Milner, Richard N., Rt. 1, Cashmere, Wash.
 Miner, Kingsley M., 210 N. 12th St., Sac City, Iowa
 Mitchell, Joseph G., Jr., Baptist Hospital, 1000 Moneno St., Pensacola, Fla.
 Moegenburg, Corrine R., 1863 E. 7th St., St. Paul
 Mohri, Judith J., 415 W. 46th Terr., Kansas City, Mo.
 Monical, Beverly B., 4489 Laclede, St. Louis
 Mooney, Lawrence P., 1606 St. Johns Ave., Billings, Mont.
 Moore, Eunice O., 9483-B Webbs Chapel Rd., Dallas, Texas

Moran, Patricia J., 2524 Saratoga Dr., Louisville, Ky.
 Moran, Richard A., PT Dept., Memorial Hospital, 3200 Noyes Ave.,
 S. E., Charleston, W. Va.
 Moriarty, Morton W., 3406-A Southern Oaks Blvd., Dallas, Texas
 Mosallert, Norman J., 1122 Thorane Blvd., San Antonio, Texas
 Mueller, Annette M., 4104 Francis, Kansas City, Kans.
 Mulnix, Rozanne, 513 N. Fowler, Meade, Kans.
 Mutschler, Elizabeth M., 304 Church Ave., Pittsburgh
 Mutter, Sylvia K., 1940 Seventh Ave., Oakland, Calif.

N

Nahlik, Sr. M. Jolendis, 1100 Bellevue Ave., St. Louis
 Nanninga, Leah R., 1116 Washington Blvd., Oak Park, Ill.
 Nash, Jeanine T., 2010 Oaklawn, La Marque, Texas
 Nathanson, Eileen, 142 E. 56th St., Brooklyn
 Natrop, Carol M., 1010 Division St., La Crosse, Wis.
 Neale, John K., Sand Springs Rd., Williamstown, Mass.
 Nelson, Donna Louise, 103 7th St., Santa Rosa, Calif.
 Nerli, Louis S., 130 Storms Ave., Jersey City, N. J.
 Nesselthaler, Jane V., 1224 Payne St., Fredericksburg, Va.
 Newcome, Doris A., 209 W. Woodland Ave., Youngstown, Ohio
 Newman, Ida M., 1630 Ford Pkwy., St. Paul
 Nicholas, Alfred E., 4 James St., Highland Park, N. J.
 Nicoll, Marjorie G., 597 Kearsage Ave., Elmhurst, Ill.
 Niehuss, Jacquelyn E., 705 Gary St., Augusta, Ga.
 Nielsen, Sandra Jean, 1452 W. Fargo Ave., Chicago
 Nies, Nan K., 118 Edison St., Wyckoff, N. J.
 Nishi, Yoko, 113 S. Occidental Blvd., Los Angeles
 Nixon, Mary G., 15701 Stone Ave., Seattle
 Noble, Barbara J., 4565 W. 3500 S., Salt Lake City
 Northrup, Vernice M., 8891 Rutherford, Detroit
 Nunke, Carol, 2505 Aqueduct Ave., New York
 Nunnery, Blanche L., 3703 Worth St., Dallas, Texas
 Nuttycombe, Elizabeth J., 2322 Lacy Lane, Richmond, Va.

O

Obirek, Joan A., 4517 S. Whipple St., Chicago
 O'Connor, Mary Patrice, 5834 S. Stony Island, Chicago
 Odenkirk, Barbara J., 604 N. Vermont, Arlington, Va.
 Offermann, Shirley V., 140 N. Broadway, Irvington, N. Y.
 Oldenburg, Douglas J., 117 S. 19th St., La Crosse, Wis.
 Oliver, Rolene A., 133 W. Tulane, Columbus, Ohio
 Olofsson, Harold W., 12211 S. Eggleston Ave., Chicago
 Olson, Barbara C., 701 20th St., Primghar, Iowa
 Osterberg, Richard D., Florence, Wis.
 Ostermeier, Barbara T., Rt. 1, Midland, Ohio
 Oswald, Dean P., 511 Finkbine Park, Iowa City
 Oxford, Patsy R., 5420 Lewis, Dallas, Texas
 Ozburn, Mary Sue, Tripler Army Hospital, Box 190, APO 438,
 San Francisco

P

Palmer, Betty June, 1205 Danube, Houston, Texas
 Parker, Ann, 4203 Wycliff, Dallas, Texas
 Parker, Deonne, 641 Sligo Ave., Silver Spring, Md.
 Parks, Travis B., 701 E. Hillcrest Rd., c/o Fantom, York, Pa.
 Parr, Carol Ann, Highland View Hospital, PT Dept., Harvard Rd.,
 Cleveland
 Pascarella, Edward A., PO Box 213, Roxboro, N. C.
 Paschall, Nancy Jo, 443 Berkshire Rd., Charlotte, N. C.
 Patterson, Clarence N., 1313 10th St., N. W., Roanoke, Va.
 Patterson, Robert E., 1056 West Ave., Lancaster, Calif.
 Patton, Nancy J., 320 S. Division, Ann Arbor, Mich.
 Pavlis, Patricia M., PT Section, Beach Pavilion BFH, Ft. Sam
 Houston, Texas
 Percy, Martha L., 69 W. Columbus St., Martinsville, Ind.
 Peik, Nancy Lee, 1808 S. 11th St., Sheboygan, Wis.
 Penn, William H., 1018 E. Wayne, South Bend, Ind.
 Pernusch, Barbara Ann, 430 John St., Kalamazoo, Mich.
 Perrine, J. Neal, 1622 Woodward Ave., Lakewood, Ohio
 Perring, Kay, Henry Street Settlement, 265 Henry St., New York
 Peters, James J., 3110 "W" St., Sacramento, Calif.
 Petersen, Marilyn J., 3606 Ames Ave., Omaha
 Peterson, Rieta J., 30 W. Portland St., Phoenix, Ariz.
 Petrykowski, Claudia A., 11320 Arrowhead Trail,
 Hales Corners, Wis.
 Petterson, Laura, Yorkleigh Apts., 1017 London Rd., Duluth, Minn.
 Phillips, Mary E., 9815 Hocher Dr., Merriam, Kans.
 Piper, Nick J., 140 E. Court, Ottumwa, Iowa
 Pletcher, Jean P., 417 Riverside Dr., New York
 Pocock, Carol Ann, 3429 Hawthorne Ave., Omaha
 Polka, Regina, 531 W. Roscoe St., Chicago
 Potash, Mary-Allen, 674 W. 161st St., New York
 Poyner, Robert N., 819 Riverdale Dr., Dallas, Texas
 Poynter, Joyce A., 17725 Franklin, Lakewood, Ohio

Pratt, Neal E., Oriole Ave., Media, Pa.
 Prettyman, Eve, 1475 N. W. 14th Ave., Miami, Fla.
 Pretzer, Arlo C., Box 243, Minot, N. D.
 Price, Janice G., 609 W. Rosewood Ct., Ontario, Calif.
 Prince, Dudley E., 1508 Madison St., Hyattsville, Md.
 Propst, Martha Ann, Box 123, USAF Hospital, Wright-Patterson
 AFB, Ohio
 Prouty, Marcia D., 343 1/2 King Ave., Columbus, Ohio
 Pruett, Herbert R., USPHS Hospital, 77 Warren St., Boston

Q

Quick, John T., 741 Melrose Ave., Iowa City

R

Rademacher, Joan, 2014 S. 3rd Ave., Maywood, Ill.
 Rahe, Laurie D., 154 Valley St., Seattle
 Raiewski, John A., 1044 Pine St., San Francisco
 Rakolta, Marie S., 410 N. Thayer, Ann Arbor, Mich.
 Ramey, Mary M., 19 Lyman St., Beverly, Mass.
 Ratch, Nancy R., 3101 Humboldt, Topeka, Kans.
 Raymond, Patricia Ann, 3922A Jamieson St., St. Louis
 Redenius, Myron E., St. Francis Hospital, Colorado Springs, Colo.
 Reid, Betty B., 10250 S. E. 21st St., Bellevue, Wash.
 Reifsnnyder, Robert N., 184 W. Durham, Philadelphia
 Reiner, Yvette M., 8001 Eastern Dr., Silver Spring, Md.
 Reynolds, Harriet N., 432 N. 15th St., Kansas City, Kans.
 Riaboken, Yaroslava, 1000 Hague Ave., St. Paul
 Ricciardi, Catherine E., 191 Trenton Ave., Paterson, N. J.
 Richardson, Barbara Anne, PT Clinic, Womack Army Hospital, Ft.
 Bragg, N. C.
 Richardson, Robert W., 1411 Poplar St., Pittsburgh
 Rickard, Virginia L., 1598 S. Third, Corvallis, Ore.
 Riesselman, Judith Ann, 1360 Monroe, Denver
 Rinde, Mary Louise, 419 Seaside Ave., Honolulu, Hawaii
 Ritchey, Henry C., 719 Cherry, N. W., Ardmore, Okla.
 Ritter, Joseph J., 356 Gardner Ave., Trenton, N. J.
 Rivers, Lois M., RD #2, Laurel Dr., Granby, Conn.
 Robb, Marcia, 751 Troy Ave., Brooklyn
 Robbers, Diann L., 1208 Maiden Lane, Pullman, Wash.
 Roberts, Patricia R., 106 E. 28th St., Cheyenne, Wyo.
 Roberts, Paul A., 4105 N. 21st St., Omaha
 Robinson, Carol P., 326 9th Ave., Seattle
 Robinson, John W., 1516 Widener Pl., Philadelphia
 Robinson, Jon R., Harlan Memorial Hospital, PM&R Dept.,
 Harlan, Ky.
 Robinson, Terry M., 1247 Danville Dr., Richardson, Texas
 Rodgers, G. Maureen, 848 Clarkson St., Denver
 Roehrs, Nancy S., 393 N. Virginia Lee Rd., E., Columbus, Ohio
 Rohlf, Sherry D., 2230 W. "O" St., Lincoln, Neb.
 Roman, Michael P., 3 E. Buttonwood St., Hazleton, Pa.
 Romine, Marilyn G., Address unknown
 Roof, Margaret S., Address unknown
 Roos, Sharon L., 1088 South St., Dalton, Mass.
 Rose, LaRea W., 807 Iola St., Aurora, Colo.
 Rose, Mavis V., 56 W. 112th, New York
 Rosen, Harriet S., 3155 Rochambeau Ave., Bronx, N. Y.
 Rosenberger, Gregory C., 1607 Monroe Ave., Evansville, Ind.
 Rosselot, Inge K., 500 Shrewsbury Ct., New Orleans
 Roughgarden, Nancy J., 419 Squaw Brook Rd., Paterson, N. J.
 Rozell, Walter V., 169 Shady Grove Dr., Riverside, Calif.
 Rozeman, Barbara P., 437 Aldine, Chicago
 Runte, Earl W., 1817 45th St., Des Moines, Iowa
 Rust, Jack R., 7580 N. W. 28th St., West Hollywood, Fla.
 Rutan, Fred M., 1902 N. E. 8th St., Gainesville, Fla.
 Ruzic, Samuel W., 1203 N. Fellows Ave., Ottumwa, Iowa
 Ryan, Doreen R., Lake Shore Rd., Plattsburgh, N. Y.

S

Saddoris, Richard G., 3418 47th, Des Moines, Iowa
 Sadowski, John R., 1 Valley St., Northampton, Mass.
 Saenz, Dale A., 1003 N. E. 10th Ave., Gainesville, Fla.
 St. Pierre, Ronald L., 2161 W. Grand Blvd., Detroit
 Sakata, Jane S., 2036 Victoria Ave., Los Angeles
 Salzman, Gladys T., 436 N. 5th St., San Jose, Calif.
 Samsel, Ruth L., 5 Dogwood Lane, Old Bridge, N. J.
 Samuels, Phyllis A., 2063 80th St., Brooklyn
 Sander, Dawna Y., Address unknown
 Santana, Edwin R., Baldorioty 1, Cabo Rojo, P. R.
 Sawatsky, James L., 121 Duncan Terr., RR 3, Newton, Kans.
 Sawyers, James M., 4603 S. Renelle Dr., Tampa, Fla.
 Scaff, Beverly E., 2065 B Mather Way, Elkins Park, Pa.
 Scaramuzza, Virginia Ann, 67 Magnolia, Goleta, Calif.
 Scharzt, Barbara E., 760 Greenwood Ave., Cincinnati
 Schaub, Judith L., 89 Powell Rd., Springfield, Pa.
 Schmidt, Ann C., 21 N. Madison, Allentown, Pa.

- Schmidt, Richard L., 1721 Kirkwood Dr., Kalamazoo, Mich.
 Schmidt, Virginia Ann, 2753 Asbury Ave., Evanston, Ill.
 Schmidt, Walter M., 265 Woodbridge Ave., Clifton Heights, Pa.
 Schnarrs, Kenneth L., 641 Pear St., Reading, Pa.
 Schneider, Paul D., 1435 N. 10th St., Beatrice, Neb.
 Schneiman, Toby Ann, 8945 Dewees St., Philadelphia
 Schober, William R., 3200 E. 8th, Des Moines, Iowa
 Schultz, Elizabeth K., Box 949, Marion, Ind.
 Schwark, Eleanor V., 1359 N. Hudson, Chicago
 Scott, Joey Lee, 407 "E" St., Beckley, W. Va.
 Scully, Rosemary M., 674 W. 161 St., New York
 Seale, Sarah G., 225 D Hillcrest Manor, Utica, N. Y.
 Seagrist, Margaret Ann, 4256 Toledo, St. Louis Park, Minn.
 Sebastian, Sally L., 800 Queen Ann, Seattle
 Setoguchi, Toshie M., 306 Parnassus Ave., San Francisco
 Sevier, Caroline A., Caddo School for Exceptional Children, 3202 Williams, Shreveport, La.
 Seymour, Elaine G., 668 Nye Circle, Radford Terr., Honolulu, Hawaii
 Shaffer, Mary O., 3740 Old William Penn Highway, Pittsburgh
 Shanks, Harold A., 328½ N. "C" St., Exeter, Calif.
 Shantz, Tracy E., 2385 3rd Ave., San Bernardino, Calif.
 Shapiro, Marcia P., Old Ford Dr., Camp Hill, RD 1, Pa.
 Shaughnessy, Mary Kay, 1613 S. Erie, Wichita, Kans.
 Shechtman, Donald Y., 313 Danny Lane, Northvale, N. J.
 Sheffer, Chester L., Jr., 6529 Holliday Rd., Richmond, Va.
 Shefts, Richard S., 2518 Farmington Rd., Peoria, Ill.
 Shehigian, Ruben B., 7807 Walnut Ave., Hammond, Ind.
 Shelton, David S., 217 Wingate Ave., S. W., Huntsville, Ala.
 Sherwood, Doris H., 741 Seward, Detroit
 Shidler, Mervin P., 5011 Jone Dr., Ft. Wayne, Ind.
 Shirkey, William L., 1251 East Ave., Elyria, Ohio
 Shoemaker, Ann L., Address unknown
 Showalter, Janet C., 6220 Jeffrey Rd., Richmond, Va.
 Shufelt, Allan R., 1222 9th St., N. E., Rochester, Minn.
 Shy, Virginia T., 4534 Parkview Pl., St. Louis
 Sieck, Margaret L., 601 Vine St., Millville, N. J.
 Siegel, Mary R., 2851 S. Herman St., Milwaukee
 Siegelman, Stanley D., 2350 E. 29th St., Brooklyn
 Sillari, Joan R., 163 Baier Ave., New Brunswick, N. J.
 Silverstein, Barbara M., 352 Maple St., New Bedford, Mass.
 Simpson, Charles C., 780 Ridge Rd., Ambridge, Pa.
 Sisson, Louise T., 2212 Strader Terr., St. Joseph, Mo.
 Skifford, Marvin, 4430 Pine St., Philadelphia
 Skelton, Mary H., 531 Brainerd Ave., St. Paul
 Skinner, Robert D., Address unknown
 Skinner, Sandra Lee, 3330 University Ave., Des Moines, Iowa
 Slade, Thomas E., 4605 S. Wellington St., Salt Lake City
 Sloth, Sarah E., 1724 S. Dale Mabry, Tampa, Fla.
 Smidt, Gary L., 129 Raritan Ave., Staten Island, N. Y.
 Smith, Alva J., Address unknown
 Smith, Barbara Anne, 822 Lawson Ave., Steubenville, Ohio
 Smith, Candace M., 3605 Baring St., Philadelphia
 Smith, Delbert J., Kalskag, Alaska
 Smith, Geraldine B., 2148 Stanford Ave., Baton Rouge, La.
 Smith, James D., 5 Merriman Rd., Kopsa Trailer Ct., Ambridge, Pa.
 Smith, Jolene R., PT Clinic, Walter Reed Hospital, Washington, D. C.
 Smith, Judith B., 1720 King Ave., Columbus, Ohio
 Smith, Lynn D., 482 E. Norwich Ave., Columbus, Ohio
 Smith, Marsha H., 1500 S. Santa Fe, Salina, Kans.
 Smith, Ruby L., 908 Spruce St., Quincy, Ill.
 Snee, Judith F., Address unknown
 Snelling, Joseph C., 1309 4th Ave., W., Birmingham, Ala.
 Soderstrom, Patsy M., 2558 Onandaga Dr., Columbus, Ohio
 Sokey, Nancy M., 348 Huntington St., Buffalo
 Sokos, Frances M., 73-24th St., Wheeling, W. Va.
 Sorkin, Alma, 434 E. 5th St., Brooklyn
 Sossong, Victor A., PM Serv., Loma Linda Hospital, Loma Linda, Calif.
 Soukup, Allen J., 920 Glendale, Newton, Kans.
 South, Bruce D., Address unknown
 Spala, Helen M., 17 Murray St., Ft. Monroe, Va.
 Sparacino, Tony J., 815 Manchester, Westchester, Ill.
 Speight, Curtis, 115 S. Second St., Greenville, Pa.
 Spencer, Marilyn M., 3206 Maury St., Richmond, Va.
 Sperandio, Martha C., USAF Hospital, Box 10334, Maxwell AFB, Ala.
 Spindler, Margaret U., Culvers Lake, Branchville, N. J.
 Spirt, Joan L., 2147 E. 17th St., Brooklyn
 Sprester, Norma J., 1501 E. 12th St., Winfield, Kans.
 Squire, Paula D., 304½ E. Adams St., Sandusky, Ohio
 Stanek, Donald W., Officers Serv. Center, Box 143, Walter Reed AH, Washington, D. C.
 Stark, Nancy S., 515 W. 46th, Kansas City, Mo.
 Steeves, Joan, Evans House, Mountaineer Hospital, Montclair, N. J.
 Steinberg, Louis H., 5953 Drexel Rd., Philadelphia
 Stenback, Jan R., 221 N. Broadmoor, Topeka, Kans.
 Stenerson, Inez E., PE Dept., Concordia College, Moorhead, Minn.
 Stephenson, Carol J., 6800 N. Baltimore, Kansas City, Mo.
 Stevens, Marilyn L., 1144 W. 29th St., Los Angeles
 Stevens, Norma J., 316 W. Park Dr., Ft. Lauderdale, Fla.
 Stewart, Elizabeth P., 2913 Chamberlayne Ave., Richmond, Va.
 Stimmel, Barry E., 7713 Carroll Ave., Takoma Park, Md.
 Stoudt, Carol M., 15235 Page Ave., Harvey, Ill.
 Stragier, Mac Donald, 608 E. 6th St., Claremont, Calif.
 Stroud, James G., Rt. 1, Harmony, N. C.
 Strumia, Lucia J., 16155 Petos Key, Detroit
 Subotnik, Joyce S., 508 Park Ave., Paterson, N. J.
 Summers, Mary W., Thomas Trailer Ct., N. Broad St., Fairborn, Ohio
 Sumner, Anne D., 3132 Meadowbrook Blvd., Cleveland Heights, Ohio
 Swanson, John A., 3127 N. Racine Ave., Chicago
 Sweetman, Charles H., Jr., 132 Berkeley Ave., Colonia, N. J.
 Swider, Donna M., 9034 Cambridge Dr., Northfield Village, Ohio
 Swift, Lucile M., 2006 W. 3rd, Bloomington, Ind.
 Sykes, Mary C., 1207 E. 60th St., Chicago
- ## T
- Tagawa, Theodore T., 2538 W. Ramona Rd., Alhambra, Calif.
 Taggart, Ann, Box 744, Luling, Texas
 Taugher, Brian D., 623 Boyd St., Oshkosh, Wis.
 Taylor, Clare T., VP-4 Navy 3867, FPO, San Francisco
 Taylor, Jill W., 221 W. 14th St., Wilmington, Del.
 Taylor, Wayne H., 1314 Park Ave., Omaha
 Teague, Patricia S., 519 Rue St. Ann, New Orleans
 Teegarden, Mary G., Westlawn P. O. Box 45, Iowa City
 Theriault, Florent A., 755 W. Bittersweet Pl., Chicago
 Thomas, Sr. M. Patricia Ann, 152-11 89th Ave., Jamaica, N. Y.
 Thompson, James T., 20901 S. Lakeshore Blvd., Euclid, Ohio
 Thompson, Joan S., Address unknown
 Thompson, M. James, 4801 Canton, Lubbock, Texas
 Thompson, Patricia H., D. T. Watson Home for Crippled Children, Leetsdale, Pa.
 Thoms, Lois E., 508 Lawrence, Ann Arbor, Mich.
 Thornburg, Noel G., 538 S. Main St., Parker, Ind.
 Tighe, Martha S., 503 W. 46th St., Kansas City, Mo.
 Toews, Julius V., 11521 Norwood, La Sierra Station, Arlington, Calif.
 Tomosawa, Francis M., 1003 N. New Hampshire Ave., Los Angeles
 Towne, Peter A., 1010 Cereal Ave., Hamilton, Ohio
 Towns, Martha J., 212 E. 6th St., Hays, Kans.
 Townsend, Ronnie E., 314 Walker St., Danville, Va.
 Trask, Irene S., 801 6th St., S. E., Minneapolis
 Tresch, Donald D., 200 S. Market St., New Athens, Ill.
 Troske, Frank H., Jr., Address unknown
 Tullis, Reeta G., 3006 Victoria Blvd., Hampton, Va.
 Turner, Fred J., 165 169th St., Hazel Crest, Ill.
 Turner, Glenn A., Jr., 314 Smith St., Gonzales, Texas
 Twist, Dulce J., 520 W. 122nd St., New York
- ## U
- Uhlhorn, Raymond W., 1806 Van Camp Ave., Omaha
 Ulrich, F. Thomas, Address unknown
 Unger, Julianna S., 30 Prospect Ave., Hackensack, N. J.
 Unser, Helen C., Morrisonville, Ill.
 Upperman, Mae B., 869 Parkwood Dr., Cleveland
 Utt, William H., 319 Broadfoot Ave., Fayetteville, N. C.
- ## V
- Van Amburgh, Roxane B., 35 Natick Ave., Warwick, R. I.
 Vanderlan, Wayne A., 502 5th Ave., Charles City, Iowa
 Vander-Meyden, Joyce, 152 Minerva St., Syracuse, N. Y.
 Van Divort, Judith P., 4707 24th, N. E., Seattle
 Van Patten, Earl L., 1436 Bonella, Walla Walla, Wash.
 Varney, Mary J., 732 N. 17th St., Milwaukee
 Vazquez, Amalia J., 348 Leonidas Aguirre 348, Mendoza, Argentina
 Vegso, Sally W., 1155 Linda Vista, Dayton, Ohio
 Vencill, D. Paul, 1279 W. 6th Ave., Eugene, Ore.
 Verdon, Nancy Ann, 1731 Vallejo St., San Francisco
 Verlee, Gwendolyn L., 1469 University Terr., Ann Arbor, Mich.
 Vespaziano, Jack L., Rt. 5, Box 237, Morgantown, W. Va.
 Vestal, Kelly J., 336 Summit Ave., Jersey City, N. J.
 Villano, Jerry J., 1811 W. 82nd Pl., Denver
 Villanueva, Elia E., 73 De Diego St., Ciales, P. R.
 Volk, Marcia L., 124 9th St., N. E., Rochester, Minn.
 Von Rump, Betty L., 9605 Twincrest Dr., Crestwood, Mo.

W

Wagner, Howard V., 707 Mallison Ave., Akron, Ohio
 Wagner, Merlee N., 1917 S. 59th Ct., Cicero, Ill.
 Waits, Donald C., 3317 Pennsylvania, Kansas City, Mo.
 Walcher, J. Ronald, 229 E. 80th, New York
 Waldschmidt, Janet C., 5665 N. Bay Ridge Ave., Milwaukee
 Walker, Hilda L., 1300 Brooks Ave., Raleigh, N. C.
 Walkinshaw, Roy M., 945 N. Vassar, Wichita, Kans.
 Walling, Cynthia Ann, 3825 S. Berkley Circle, Cincinnati
 Walter, Ella M., 235 S. 44th St., Philadelphia
 Walters, Michael N., 116 Pomeroy, Pasadena, Texas
 Wandel, Jerome L., Jr., 1000 Garden Pl., Glenshaw, Pa.
 Ward, Mary G., 336 Summit Ave., Jersey City, N. J.
 Warner, Sallie S., 1005 Little Bay Ave., Norfolk, Va.
 Warren, Patricia, 4141 Francis St., Kansas City, Kans.
 Waterman, Nancy W., 1116 Front St., Vestal, N. Y.
 Waters, Clarence P., 808 51st St., Great Falls, Mont.
 Weber, Janell K., 415 Erie St., S. E., Minneapolis
 Weber, Mary Ellen, 3604 Plyers Mill Rd., Kensington, Md.
 Weckstein, Lillian N., 42 Coolidge Rd., Maplewood, N. J.
 Weigly, Lillian D., 5510 N. Rosslyn Ave., Indianapolis
 Weiland, Louis H., Address unknown
 Weinberg, Mary Patricia, 215 Crespi Ave., Exeter, Calif.
 Weir, Lavonne C., 3592 Elmwood Dr., Riverside, Calif.
 Weiskopf, Albert, 180 Fairlawn Ave., Albany, N. Y.
 Weiss, Paul, 623-5 E. 5th St., Brooklyn
 Wellenstein, Mary K., Clarkson Hospital, PT Dept., Omaha
 Wendeborn, J. D., Rt. 1, Sagerton, Texas
 Wennerberg, Ruth C., 48-17 186th St., Flushing, N. Y.
 Wesotsky, Olga, 233 E. 80th St., New York
 Wessman, Henry C., 328 Crompt St., Crookston, Minn.
 Westenber, Beverly P., 303 E. Edgewater St., Box 67, Cambria, Wis.
 Weston, Mary Jane, 5610 Martel St., Dallas, Texas
 Whipkey, Noel G., 330 Ohio River Blvd. Plaza, Sewickley, Pa.
 Whitaker, Joyce L., 13 Grove St., Boston
 White, Wanda B., 1140 Ash, Denver
 Whiting, Gwendolyn L., 5408 Kalmia Dr., Orlando, Fla.
 Whitney, Sue W., 330 Ohio River Blvd., Sewickley, Pa.
 Whitten, Virginia W., 2417 Dozier Dr., Tallahassee, Fla.
 Wiebke, Marilyn L., 4141 Francis, Kansas City, Kans.

Wilke, Dorothy R., 1697 Wyandotte Rd., Columbus, Ohio
 Williams, Carol Lee, 2248 Augusta Rd., Greenville, S. C.
 Williams, Heather A., 1854 1/2 12th St., Santa Monica, Calif.
 Williams, Lloyd D., 2900 E. College, Boulder, Colo.
 Williams, Peggy A., 4141 Francis St., Kansas City, Kans.
 Williams, Rob-Roy, 1007 S. Watts Ave., Sioux Falls, S. D.
 Wilson, Mildred L., 105 W. Lancaster Ave., Downingtown, Pa.
 Wiseman, Nancy Lee, 3350 S. Wakefield St., Arlington, Va.
 Witt, James L., 445 Fautoute Ave., Roselle Park, N. J.
 Wolfers, June H., 654 Oakside Ave., Redwood City, Calif.
 Womack, Phala A., 3908 Byers, Ft. Worth, Texas
 Womack, William F., 1012 W. 8th St., Chester, Pa.
 Woodhouse, Carol K., 105 N. Clinton St., Iowa City
 Woodley, Aminta J., 5543 Morton St., Philadelphia
 Wyatt, David L., Box 1559, Lackland AFB Hospital, San Antonio, Texas
 Wyatt, Katherine V., Slingerlands R. D., N. Y.
 Wythe, Gillis, Jr., 918 Duval, Temple, Texas

Y

Yalch, Joseph P., 2249 Barrington Rd., Bethlehem, Pa.
 Yarbrough, Patricia, 855 Briarcliff Rd., N. E., Atlanta, Ga.
 Yatchum, John G., 411 Stock St., Hanover, Pa.
 Yerger, Barbara Ann, Montebello State Hospital, 2201 Argonne Dr., Baltimore, Md.
 Young, Elizabeth C., 1118 1st St., N. W., Rochester, Minn.
 Young, Gordon O., 314 E. 157th St., Harvey, Ill.
 Yunker, Ann J., 924 Custer Dr., Toledo, Ohio
 Yurko, Louise M., 1802 Cornell Ave., Richmond, Va.

Z

Zahnd, Mary J., 315 E. 48th, Kansas City, Mo.
 Zhe, Carolyn R., 308 King's Rd., Schenectady, N. Y.
 Zigler, Beverly Ann, 241 6th Ave., New York
 Zimmerman, Joseph E., 407 'E' St., Beckley, W. Va.
 Zupan, Sr. M. Dorine, 1004 N. Tenth St., Milwaukee
 Zwahlen, Nina A., 2140 Grand Ave., Des Moines, Iowa
 Zylstra, Marvin K., 31 Truman Blvd., Oakland, N. J.

AMERICAN REGISTRY OF PHYSICAL THERAPISTS
GEOGRAPHIC SECTION
DIRECTORY SUPPLEMENT S-1
 (Complete address appears in alphabetic section, Page 293)

ALABAMA

Bowlen, D., Mobile—'60
 Boze, C. R., Anniston—'60
 Cannon, V. S., Montgomery—'60
 Cook, G. W., Jr., Anniston—'59
 Shelton, D. S., Huntsville—'59
 Snelling, J. C., Birmingham—'58
 Sperandio, M. C., Maxwell AFB—'58

ALASKA

Smith, D. J., Kalslag—'60

ARIZONA

Gould, B. J., Phoenix—'59
 Julian, J. A., Phoenix—'58
 Julian, N. R., Phoenix—'58
 Peterson, R. J., Phoenix—'60

ARKANSAS

Cravens, M. W., Little Rock—'60
 Johnson, O. Jr., State College—'59

CALIFORNIA

Berriman, D. R., Arcadia—'58
 Blankenship, J. A., Carmichael—'58
 Blumenfeld, D. M., Sacramento—'58
 Brayshaw, G. D., Loma Linda—'61
 Brown, M., San Francisco—'58

Buzard, L. A., Long Beach—'59
 Clemenson, S. A.,
 Presidio of San Francisco—'59
 Cooper, E. S., San Diego—'60
 Cornell, E. J., San Rafael—'60
 Davis, R. G., Long Beach—'60
 Dedeker, R. M., Los Angeles—'60
 Diringier, E. C., San Francisco—'60
 Ellis, N. L., Hollywood—'60
 Enomoto, R. T., Los Angeles—'58
 Field, K. C., Santa Ana—'60
 Garrity, K. H., Long Beach—'59
 Gottdank, M. B., Costa Mesa—'58
 Gracial, L. S., Covina—'47
 Gray, B. A., Corte Madera—'59
 Greenwald, N. L., Long Beach—'60
 Habenicht, C. W., Arlington—'61
 Haffner, E. L., Oxnard—'60
 Hardy, P. O., Loma Linda—'61
 Harrison, R. R., Riverside—'60
 Hawkes, A. M., Santa Barbara—'59
 Hubbs, M. E., Santa Rosa—'60
 Isaacson, G. K., San Francisco—'58
 Jensen, I. R., Bakersfield—'58
 Kaczorowski, J. A., Santa Monica—'59
 Knox, W. L., Modesto—'58
 Kow, K. C., Los Angeles—'60
 Kuivinen, B. C., Oakland—'60

Lane, L. A., Oakland—'60
 Lang, R. D., Loma Linda—'60
 Leonard, D. G., Los Angeles—'59
 Lynch, G. M., San Diego—'60
 Maniscalco, L., Daly City—'60
 Masato, J. J., Los Angeles—'58
 Mehus, S. F., Orinda—'60
 Miller, M., Porterville—'59
 Mutter, S. K., Oakland—'60
 Nelson, D. L., Santa Rosa—'60
 Nishi, Y., Los Angeles—'60
 Ozburn, M. S., San Francisco—'58
 Patterson, R. E., Lancaster—'60
 Peters, J. J., Sacramento—'60
 Price, J. G., Ontario—'59
 Raiewski, J. A., San Francisco—'60
 Rozell, W. V., Riverside—'60
 Sakata, J. S., Los Angeles—'58
 Salzman, G. T., San Jose—'59
 Scaramuzza, V. A., Goleta—'60
 Setoguchi, T. M., San Francisco—'60
 Shanks, H. A., Exeter—'60
 Shantz, T. E., San Bernardino—'60
 Sossong, V. A., Loma Linda—'61
 Stevens, M. L., Los Angeles—'58
 Stragier, M. D., Claremont—'60
 Tagawa, T. T., Alhambra—'61
 Taylor, C. T., San Francisco—'60

Toews, J. V., Arlington—'60
 Tomosawa, F. M., Los Angeles—'60
 Verdon, N. A., San Francisco—'59
 Weinberg, M. P., Exeter—'60
 Weir, L. C., Riverside—'60
 Williams, H. A., Santa Monica—'59
 Wolfers, J. H., Redwood City—'60

COLORADO

Aten, D. W., Colorado Springs—'60
 Beam, T. M., Greeley—'60
 Brown, D. R., Denver—'60
 Chabas, J. M., Denver—'60
 Clement, J. P., Denver—'59
 Coles, S. N., Denver—'59
 Deibler, P. C., Pueblo—'59
 Dontigny, R. L., Colorado Springs—'58
 Friedmeyer, M. S., Lowrey AFB—'59
 Goodwin, J. R., Denver—'60
 Hall, M. S., Ft. Carson—'58
 Harrison, D. J., Ft. Collins—'59
 Isaacs, L. E., Denver—'58
 Laulainen, J. A., Denver—'60
 Lee, B. H., Brush—'60
 Lewis, M. A., Denver—'58
 Mattox, D. L., Denver—'60
 Redenius, M. E., Colorado Springs—'59
 Riesselman, J. A., Denver—'60
 Rodgers, G. M., Denver—'59
 Rose, L. W., Aurora—'58
 Villano, J. J., Denver—'58
 White, W. B., Denver—'60
 Williams, L. D., Boulder—'59

CONNECTICUT

Casey, R. G., Bristol—'60
 Cleary, Sr., M. T. P., Trumbull—'60
 Haley, A. T., New Haven—'59
 Hemenway, V. R., West Haven—'59
 Jaekle, R. C., Seymour—'60
 Rivers, L. M., Granby—'51

DELAWARE

Taylor, J. W., Wilmington—'58

DISTRICT OF COLUMBIA

Billion, C. D., Washington—'58
 Falor, S. L., Washington—'60
 Hamilton, D. W., Washington—'58
 Hassan, R. O., Washington—'50
 Hessler, D. J., Washington—'60
 Jackson, C. E., Washington—'60
 McGee, P. P., Washington—'58
 Smith, J. R., Washington—'60
 Stanek, D. W., Washington—'59

FLORIDA

Amundson, L. D., West Palm Beach—'60
 Barish, S., Coral Gables—'60
 Cone, J. W., Clearwater—'58
 Durrance, J. B., Tampa—'58
 Goodwin, M. K., Jacksonville—'60
 Hanes, M. J., Miami—'58
 Knapton, R. G., West Palm Beach—'60
 Kressley, N. W., MacDill AFB—'60
 Kurras, R. A., Miami—'59
 Mitchell, J. G., Jr., Pensacola—'61
 Prettyman, E., Miami—'43
 Rust, J. R., West Hollywood—'58
 Rutan, F. M., Gainesville—'59
 Saenz, D. A., Gainesville—'60
 Sawyers, J. M., Tampa—'58
 Slohn, S. E., Tampa—'59
 Stevens, N. J., Ft. Lauderdale—'61
 Whiting, G. L., Orlando—'55
 Whitten, V. W., Tallahassee—'47

GEORGIA

Burgamy, W. R., Jr., Macon—'59
 Coulter, J. J., Augusta—'58
 Elsea, N. K., Atlanta—'59
 Haines, J., Atlanta—'60
 Niehuss, J. E., Augusta—'59
 Yarbrough, P., Atlanta—'60

HAWAII

Fuchigami, D. Y., Wahiawa—'59
 Rinde, M. L., Honolulu—'59
 Seymour, E. G., Honolulu—'60

ILLINOIS

Bacon, M. E., Chicago—'60
 Barger, S. L., Moline—'59
 Beardsley, J. C., Rock Island—'58
 Berger, W. H., Highland Park—'60
 Bigstaff, M. M., Chicago—'60
 Bondurant, J. K., Chicago—'59
 Boyd, D. B., Aurora—'58
 Boyer, C. L., Chicago—'59
 Buggs, U. A., Jr., Chicago—'59
 Clemons, B. V., Peoria—'59
 Cohen, G., Chicago—'59
 Cowan, H. L., Zion—'58
 Davison, R. L., Rantoul—'58
 Day, B. H., Libertyville—'60
 deLaVan, P. L., Oak Park—'59
 Dorn, C. G., Decatur—'60
 Gast, W. L., Rockford—'60
 Gay, M. R., Springfield—'58
 Goldberg, C. J., Highland Park—'60
 Hauser, C. A., Winnetka—'58
 Irvin, R. F., Normal—'58
 Jacobson, J. C., Chicago—'59
 Jaeger, R. C., Moline—'60
 Karnuth, H. A., Downers Grove—'60
 Klibanski, M., Chicago—'58
 Knoerzer, N. L., Chicago—'60
 Lystad, A. F., Evanston—'59
 Mandel, L. S., Chicago—'58
 McCallum, A. M., Champaign—'59
 McCarthy, J. L., Oak Lawn—'59
 McGinnis, M. C., Urbana—'60
 Nanninga, L. R., Oak Park—'59
 Nicoll, M. G., Elmhurst—'60
 Nielsen, S. J., Chicago—'58
 Obirek, J. A., Chicago—'60
 O'Connor, M. P., Chicago—'59
 Olofsson, H. W., Chicago—'60
 Polka, R., Chicago—'58
 Rademacher, J., Maywood—'58
 Rozeman, B. P., Chicago—'59
 Schmidt, V. A., Evanston—'60
 Schwark, E. V., Chicago—'60
 Shefts, R. S., Peoria—'60
 Smith, R. L., Quincy—'58
 Sparacino, T. J., Westchester—'60
 Stoudt, C. M., Harvey—'59
 Swanson, J. A., Chicago—'60
 Sykes, M. C., Chicago—'58
 Thierault, F. A., Chicago—'61
 Tresch, D. D., New Athens—'59
 Turner, F. J., Hazel Crest—'59
 Unser, H. C., Morrisonville—'60
 Wagner, M. N., Cicero—'59
 Young, G. O., Harvey—'60

INDIANA

Avery, J. L., Cutler—'60
 Badell, J., Jr., Indianapolis—'58
 Belzile, G., Terre Haute—'59
 Cooper, S. B., Evansville—'59
 Earl, E. F., South Bend—'58
 Golden, W. W., Elkhart—'60
 Helms, M. D., Terre Haute—'59
 McClatchie, J. G., Lafayette—'59
 McKinlay, F. D., Michigan City—'48
 Pearcy, M. L., Martinsville—'52
 Penn, W. H., South Bend—'58
 Rosenberger, G. C., Evansville—'59
 Schultz, E. K., Marion—'60
 Shegihan, R. B., Hammond—'60
 Shidler, M. P., Ft. Wayne—'60
 Swift, L. M., Bloomington—'44
 Thornburg, N. G., Parker—'58
 Weigly, L. D., Indianapolis—'58

IOWA

Bachli, R. G., Jr., Ft. Madison—'60
 Baker, K. L., Iowa City—'59
 Berglund, B. E., Iowa City—'58
 Calabrese, F., Des Moines—'60
 Capper, S. L., Iowa City—'60
 Clemens, L. R., Dubuque—'60
 Dahm, J. P., Des Moines—'58
 Davison, R. J., New London—'60
 Dunham, D. D., Des Moines—'60
 Fairchild, R. B., Coralville—'60

Feye, M. A., Dubuque—'58
 Hannah, P. F., Iowa City—'59
 Hass, S. A., Sac City—'60
 Hazelton, F. T., Cedar Rapids—'59
 Hoppens, N. S., Perry—'58
 Kelsen, R. R., Des Moines—'59
 Kirkegaard, P. F., Sioux City—'59
 Larsen, S. Z., Sioux City—'60
 Lofgren, S. L., Des Moines—'60
 Long, R. W., Des Moines—'60
 Miner, K. M., Sac City—'60
 Olson, B. C., Primghar—'60
 Oswald, D. P., Iowa City—'60
 Piper, N. J., Ottumwa—'58
 Quick, J. T., Iowa City—'60
 Runte, E. W., Des Moines—'60
 Ruzic, S. W., Ottumwa—'60
 Saddoris, R. G., Des Moines—'60
 Schober, W. R., Des Moines—'60
 Skinner, S. L., Des Moines—'59
 Teegarden, M. G., Iowa City—'60
 Vanderlan, W. A., Charles City—'60
 Woodhouse, C. K., Iowa City—'60
 Zwahlen, N. A., Des Moines—'60

KANSAS

Cohn, A. J., Lawrence—'60
 Derrick, B., Ft. Riley—'59
 Dicks, D., Prairie Village—'60
 Evans, N. C., Kansas City—'59
 Foerster, J. C., Lawrence—'58
 Ford, B. C., Lucas—'60
 Glass, S. H., Overland Park—'59
 Harnar, J., Lawrence—'60
 Hirsch, J., Goodland—'60
 Howard, T. L., Hutchinson—'59
 Jeffrey, J. A., Kansas City—'60
 Kendall, D. D., Kansas City—'60
 Miller, J. K., Leavenworth—'60
 Mueller, A. M., Kansas City—'61
 Mulinix, R., Meade—'60
 Phillips, M. E., Merriam—'60
 Ratch, N. R., Topeka—'59
 Reynolds, H. N., Kansas City—'60
 Sawatsky, J. L., Newton—'60
 Shaughnessy, M. K., Wichita—'59
 Smith, M. H., Salina—'58
 Soukup, A. J., Newton—'58
 Sprenger, N. J., Winfield—'61
 Stenback, J. R., Topeka—'60
 Towns, M. J., Hays—'58
 Walkinshaw, R. M., Wichita—'59
 Warren, P., Kansas City—'60
 Wiebke, M. L., Kansas City—'60
 Williams, P. A., Kansas City—'60

KENTUCKY

Brown, M. I., Ft. Knox—'58
 Campeau, T. J., Berea—'60
 Fitzgibbon, V. L., Louisville—'58
 Harris, M. A., Louisville—'60
 Haws, C. F., Jr., Paducah—'60
 Houser, R. D., Paducah—'60
 Lamar, P. A., Lexington—'58
 Moran, P. J., Louisville—'59
 Robinson, J. R., Harlan—'59

LOUISIANA

Blankenship, N. L., New Orleans—'59
 Cline, P. D., New Orleans—'58
 Daly, H. N., Lafayette—'59
 Elshree, N. H., New Orleans—'60
 Fontenot, D. M., Lake Charles—'60
 Fuller, C. K., New Orleans—'59
 Rossclot, I. K., New Orleans—'59
 Sevier, C. A., Shreveport—'58
 Smith, G. B., Baton Rouge—'59
 Teague, P. S., New Orleans—'59

MARYLAND

Becraft, J. E., Silver Spring—'59
 Custis, S. W., Mt. Rainier—'60
 Fenton, M. E., Silver Spring—'60
 Getz, L. A., Reisterstown—'59
 Lang, W. F., Salisbury—'58
 Lucas, M. E., Silver Spring—'60
 Mazzacone, R. S., Baltimore—'58

Mikush, D. C., Baltimore—'58
 Parker, D., Silver Spring—'60
 Prince, D. E., Hyattsville—'52
 Reiner, Y. M., Silver Spring—'58
 Stimmel, B. E., Takoma Park—'60
 Weber, M. E., Kensington—'58
 Yerger, B. A., Baltimore—'58

MASSACHUSETTS

Aarestad, D. M., New Bedford—'59
 Ackerman, T., Springfield—'58
 Ault, M. M., Waban—'60
 Dorsey, J. A., Jr., Andover—'59
 Dukakis, O., Arlington—'53
 Hill, J. C., Milton—'60
 Hochberg, N. J., Chestnut Hill—'59
 Johnston, B. B., Cambridge—'59
 Maeder, B. Z., Holliston—'50
 Neale, J. K., Williamstown—'59
 Pruett, H. R., Boston—'60
 Ramey, M. M., Beverly—'60
 Roos, S. L., Dalton—'58
 Sadowski, J. R., Northampton—'58
 Silverstein, B. M., New Bedford—'59
 Whitaker, J. I., Boston—'60

MICHIGAN

Allen, J. K., Wyandotte—'60
 Austin, L. A., Laingsburg—'59
 Baker, J. A., Ann Arbor—'60
 Banwell, R. G., Flint—'58
 Beger, V. J., Detroit—'59
 Burgette, P. A., Detroit—'60
 Chepey, F. T., Dearborn—'58
 Clark, D. C., Ann Arbor—'60
 Cooper, E. B., Detroit—'59
 Exelby, C. A., Ypsilanti—'59
 Figley, B. C., Midland—'60
 Fox, G. J., Ann Arbor—'59
 Guilford, J. A., Detroit—'60
 Hein, S. L., Dearborn—'60
 Howe, S. S., Ann Arbor—'59
 Huey, L. M., Ann Arbor—'59
 Hyma, B. M., Ann Arbor—'59
 Jardine, S. A., Richmond—'59
 Kamenshek, D. M., Grand Rapids—'59
 Kasten, W. R., Flint—'59
 Kazmierczak, C. B., Ann Arbor—'59
 Koch, J. A., Dearborn—'59
 Leffler, T. F., Jr., Lambertville—'58
 Link, J. R., Detroit—'59
 Locker, G. R., Ann Arbor—'60
 Meyer, E. R., Okemos—'58
 Northrup, V. M., Detroit—'59
 Patton, N. J., Ann Arbor—'58
 Pernusch, B. A., Kalamazoo—'61
 Rakolta, M. S., Ann Arbor—'60
 St. Pierre, R. L., Detroit—'60
 Schmidt, R. L., Kalamazoo—'58
 Sherwood, D. H., Detroit—'60
 Strumia, L. J., Detroit—'59
 Thoms, L. E., Ann Arbor—'61
 Verlee, G. L., Ann Arbor—'59

MINNESOTA

Anderson, K. J., Minneapolis—'59
 Anderson, R. P., St. Paul—'59
 Anger, R. E., Minneapolis—'60
 Barney, S. C., Minneapolis—'59
 Brown, M. A., Minneapolis—'59
 Cichosz, L. T., Duluth—'59
 Clayson, S. J., St. Paul—'60
 Debevec, D. F., Eveleth—'60
 Domy, M. J., Waseca—'60
 Duston, D. L., Rochester—'59
 Erickson, B. A., Rochester—'59
 Farrell, C. A., St. Paul—'60
 Garrett, T. R., Rochester—'60
 Getzel, R. M., Minneapolis—'60
 Gregerson, K., Minneapolis—'59
 Hammer, E. A., Minneapolis—'59
 Hense, T. E., Minneapolis—'60
 Higgins, J. J., Minneapolis—'60
 Howalt, G. M., Stillwater—'60
 Johnson, M. E., Minneapolis—'59
 Johnson, R. D., Virginia—'59
 Kempenich, Sr. M. V., Little Falls—'59

Kleszyk, K. A., Rochester—'60
 Kubes, B. F., Austin—'59
 Kunerth, J. M., Minneapolis—'60
 LaDuke, M. J., St. Paul—'60
 Lalim, A., Minneapolis—'59
 Lensch, R. M., Rochester—'60
 Manders, S. J., St. Paul—'59
 Millsop, C. A., Minneapolis—'60
 Moegenburg, C. R., St. Paul—'59
 Newman, I. M., St. Paul—'60
 Peterson, L., Duluth—'58
 Riaboken, Y., St. Paul—'60
 Seaquist, M. A., St. Louis Park—'59
 Shufelt, A. R., Rochester—'59
 Skelton, M. H., St. Paul—'59
 Stenerson, I. E., Moorhead—'59
 Trask, I. S., Minneapolis—'61
 Volk, M. L., Rochester—'59
 Weber, J. K., Minneapolis—'59
 Wessman, H. C., Crookston—'59
 Young, E. C., Rochester—'60

MISSISSIPPI

McMichael, G. L., Columbus—'59

MISSOURI

Averill, M. L., Clayton—'59
 Belko, A. S., St. Louis—'40
 Bowman, D. L., Kansas City—'60
 Boyer, M. O., Kansas City—'61
 Brice, F. J., Springfield—'60
 Courtal, D. C., Maplewood—'60
 Creamer, A. E., Springfield—'59
 Dahm, J. F., Richmond Heights—'60
 Evans, M. A., Kansas City—'59
 Feldman, A., St. Charles—'59
 Foote, M. E., Kansas City—'60
 Frenzel, Sr. S. M., St. Louis—'60
 Goin, P. A., Springfield—'59
 Harmon, K. A., St. Louis—'60
 Hartenbach, S. E., Creve Coeur—'60
 Holzum, Sr. G. M., St. Louis—'59
 Jacoby, P. N., Maplewood—'59
 Jarrell, B. J., Kansas City—'60
 Johnson, I. J., St. Louis—'60
 Johnson, M. G., Kansas City—'59
 Klotz, L. E., Columbia—'59
 Koellner, M. A., Brentwood—'59
 Kuehl, J. G., Webster Groves—'58
 Lied, J. E., Kirkwood—'60
 Martin, G., Marshall—'58
 Maune, S. D., Columbia—'60
 Medley, C. J., Kansas City—'60
 Mohri, J. J., Kansas City—'60
 Monical, B. B., St. Louis—'60
 Nahlik, Sr. M. J., St. Louis—'60
 Raymond, P. A., St. Louis—'60
 Shy, V. T., St. Louis—'60
 Sisson, L. T., St. Joseph—'60
 Stark, N. S., Kansas City—'59
 Stephenson, C. J., Kansas City—'60
 Tighe, M. S., Kansas City—'60
 Von Rump, B. L., Crestwood—'58
 Waits, D. C., Kansas City—'60
 Zahnd, M. J., Kansas City—'60

MONTANA

Bagley, P. A., Butte—'59
 Harbine, P. E., Missoula—'58
 Keller, P. J., Bozeman—'60
 Mooney, L. P., Billings—'59
 Waters, C. P., Great Falls—'60

NEBRASKA

Beyersdorf, M. A., Omaha—'60
 Breed, R. J., Omaha—'59
 Duhart, F. R., Omaha—'58
 Ellingham, C. T., Omaha—'58
 Ellis, R. H., Alliance—'58
 Garman, V. R., Omaha—'58
 Hammond, C. W., Potter—'59
 Petersen, M. J., Omaha—'60
 Pockock, C. A., Omaha—'60
 Roberts, P. A., Omaha—'60
 Rohlf, S. D., Lincoln—'60
 Schneider, P. D., Beatrice—'59

Taylor, W. H., Omaha—'60
 Uhlhorn, R. W., Omaha—'60
 Wellenstein, M. K., Omaha—'59

NEVADA

Baker, H. F., Reno—'60
NEW HAMPSHIRE
 Brewer, J. C., Plymouth—'55
 Fifer, B. L., Greenfield—'58

NEW JERSEY

Borbe, H. G., Maplewood—'58
 Burr, A. A., Jr., Montclair—'61
 Cherry, G. E., East Orange—'60
 Cobb, D., Boonton—'58
 Corryell, J. F., Atlantic City—'59
 Crisci, D. R., Asbury Park—'58
 Deering, E. J., Haddonfield—'58
 Delett, R. J., Haddonfield—'60
 Epps, W. H., Newark—'58
 Everett, M. A., Trenton—'58
 Fair, M. A., East Orange—'57
 Frank, A. J., Leonia—'59
 Gibson, J. C., Madison—'60
 Jolly, M. E., Bloomfield—'58
 Kovalchik, B. S., Somerville—'59
 Levine, R. H., North Brunswick—'60
 Nerli, L. S., Jersey City—'58
 Nicholas, A. E., Highland Park—'58
 Nies, N. K., Wyckoff—'50
 Ricciardi, C. E., Paterson—'61
 Ritter, J. J., Trenton—'60
 Roughgarden, N. J., Paterson—'59
 Samsel, R. L., Old Bridge—'59
 Shechtman, D. Y., Northvale—'58
 Sieck, M. L., Millville—'60
 Sillari, J. R., New Brunswick—'58
 Spindler, M. U., Branchville—'60
 Steeves, J., Montclair—'59
 Subotnik, J. S., Paterson—'59
 Sweetman, C. H., Jr., Colonia—'60
 Unger, J. S., Hackensack—'59
 Vestal, K. J., Jersey City—'59
 Ward, M. G., Jersey City—'59
 Weckstein, L. N., Maplewood—'37
 Witt, J. L., Roselle Park—'60
 Zylstra, M. K., Oakland—'58

NEW MEXICO

Boling, T. H., Albuquerque—'59
 Dohnanyi, N. H., Albuquerque—'60
 Haskew, N. M., Albuquerque—'58
 McDaniel, J. H., Albuquerque—'58

NEW YORK

Adams, E. R., Jr., East Aurora—'60
 Adar, U., Bronx—'59
 Adler, S., Brooklyn—'60
 Agate, M. A., New York—'58
 Andersson, L. G., New Rochelle—'60
 Blazi, L. B., New York—'60
 Boscolo, R. C., Stony Point—'55
 Bowman, S. Y., New York—'59
 Brady, G. J., West Haverstraw—'58
 Brenner, A. C., Buffalo—'59
 Brown, E., East Rockaway—'58
 Buechner, K. M., Brooklyn—'58
 Byron, M. A., New York—'59
 Dantzer, B. P., New York—'58
 DeChamplain, E. F., New York—'58
 Devine, R. W., Pleasantville—'60
 Edmonds, R. H., Buffalo—'59
 Edwards, E. A., New York—'58
 Foti, J. F., Watervliet—'59
 Gastaldo-Brac, N., Flushing—'60
 Goldstein, M. W., Brooklyn—'60
 Gordon, L. G., Woodmere—'59
 Grodnik, N. J., New York—'58
 Grohe, J. M., Kew Gardens—'59
 Gunter, P. E., Bronx—'59
 Hauser, M. S., New York—'60
 Heath, B. W., New York—'58
 Izzo, P. J., East Norwich—'60
 Kaplan, F. B., Brooklyn—'58
 Kerchner, J. C., Albany—'60
 Krum, P. B., Niagara Falls—'58
 Kurashige, M. C., Brooklyn—'60

Larson, A. U., Sunmount—'58
 Levitan, J., New York—'59
 Lubin, L., Howard Beach—'58
 Lupi, F. A., Utica—'58
 MacIntosh, M. W., New York—'59
 Maiseed, P. E., West Haverstraw—'60
 Marcus, J. I., Brooklyn—'59
 Nathanson, E., Brooklyn—'60
 Nunke, C., New York—'58
 Offermann, S. V., Irvington—'58
 Perring, K., New York—'60
 Pletcher, J. P., New York—'58
 Potash, M., New York—'58
 Robb, M., Brooklyn—'59
 Rose, M. V., New York—'58
 Rosen, H. S., Bronx—'60
 Ryan, D. R., Plattsburgh—'58
 Samuels, P. A., Brooklyn—'60
 Scully, R. M., New York—'58
 Seale, S. G., Utica—'59
 Siegelman, S. D., Brooklyn—'59
 Smidt, G. L., Staten Island—'60
 Sokey, N. M., Buffalo—'60
 Sorkin, A., Brooklyn—'60
 Spin, J. L., Brooklyn—'60
 Thomas, Sr. M. P. A., Jamaica—'60
 Twist, D. R., New York—'60
 Vander-Meyden, J., Syracuse—'59
 Walcher, J. R., New York—'59
 Waterman, N. W., Vestal—'58
 Weiskopf, A., Albany—'58
 Weiss, P., Brooklyn—'58
 Wennerberg, R. C., Flushing—'59
 Wesotsky, O., New York—'58
 Wyatt, K. V., Slingerlands—'55
 Zhe, C. R., Schenectady—'60
 Zigler, B. A., New York—'60

NORTH CAROLINA

Clapper, T. D., Durham—'60
 Daniels, C. F., Durham—'58
 Federchuck, R. A., Durham—'60
 Miller, M. A., Wilmington—'60
 Pascarella, E. A., Roxboro—'59
 Paschall, N. J., Charlotte—'58
 Richardson, B. A., Ft. Bragg—'60
 Stroud, J. G., Harmony—'60
 Utt, W. H., Fayetteville—'60
 Walker, H. L., Raleigh—'60

NORTH DAKOTA

Anderson, D. A., Fargo—'59
 Berkeland, H. B., Fargo—'60
 Carter, N. L., Jamestown—'59
 Goodman, J. G., Fargo—'59
 Groh, P. N., Minot—'60
 Humphrey, L. D., Fargo—'59
 Pretzer, A. C., Minot—'58

OHIO

Almone, J. A., Dayton—'60
 Argenta, C. N., Middletown—'60
 Auber, I. L., Wright-Patterson AFB—'60
 Barrett, M. L., Marietta—'59
 Berger, D. H., Circleville—'60
 Bier, D. H., Dayton—'59
 Bieseker, J. L., Dayton—'60
 Black, B. A., Youngstown—'58
 Blanc, A. M., Euclid—'60
 Blaustein, M. G., Trotwood—'59
 Bradley, V. A., Cincinnati—'58
 Butzberger, R. P., Westlake—'59
 Caldwell, K. R., Cincinnati—'59
 Canan, B. C., Lakewood—'58
 Cheuvront, L. M., Cleveland—'60
 Colananni, G. M., Cleveland—'60
 Connelly, D. P., Toledo—'60
 Crowl, L. C., Columbus—'58
 Dahl, L. M., Columbus—'58
 Davis, M. L., Akron—'60
 Dennison, G. R., Columbus—'60
 Diment, N. M., Columbus—'60
 Dobrin, M., Cleveland Heights—'60
 Dombrowsky, R. A., Cleveland—'58
 Dowell, D. J., Massillon—'60
 Dubiel, N. J., Gambier—'58
 Edgington, D. L., Cleveland—'60

Edwards, M. L., Dayton—'60
 Eliopoulos, C. G., Cincinnati—'60
 Evans, D. W., Cleveland—'60
 Fidler, L. P., Cincinnati—'60
 Fordyce, E. R., Cleveland—'58
 Franks, B. R., Columbus—'58
 Fyffe, B. A., Springfield—'58
 Gay, A. S., Cleveland—'59
 Glick, J. C., Steubenville—'60
 Glitzner, A. K., Toledo—'59
 Gothot, A. M., Cleveland—'58
 Greenwald, N. F., Columbus—'60
 Greenwood, R. R., Middletown—'58
 Halinski, W. L., Cleveland—'60
 Hall, L. M., Columbus—'60
 Hayes, S. M., Cleveland—'60
 Heidinger, P. K., East Cleveland—'58
 Hellewell, R. R., Kent—'60
 Hirsch, M. K., South Euclid—'60
 Holt, E. S., Marion—'58
 Hosmer, N. E., Louisville—'58
 Houston, B. R., Massillon—'60
 Huber, C. L., Akron—'60
 Hummer, W. G., Conneaut—'58
 James, C. A., Akron—'60
 James, D. D., Tallmadge—'60
 Janos, M. A., East Cleveland—'60
 Johanni, B. A., Cleveland—'60
 Jones, D. G., Newark—'58
 Kagler, G. W., Cincinnati—'60
 Kassell, J., Columbus—'60
 Keller, J., Cleveland—'60
 Kennedy, G. J., Columbus—'58
 Kulick, J. F., Columbus—'60
 Lee, M. U., Dayton—'60
 Loehr, F. E., East Cleveland—'60
 Lytle, J. F., Cleveland—'60
 Martini, E. D., Cincinnati—'60
 Matson, V. D., Dayton—'60
 McBroom, G. G., Columbus—'60
 Newcome, D. A., Youngstown—'59
 Oliver, R. A., Columbus—'58
 Ostermeier, B. T., Midland—'58
 Parr, C. A., Cleveland—'58
 Perrine, J. N., Lakewood—'60
 Poynter, J. A., Lakewood—'58
 Probst, M. A., Wright-Patterson AFB—'60
 Prouty, M. D., Columbus—'60
 Roehrs, N. S., Columbus—'60
 Scharz, B. E., Cincinnati—'60
 Shirkey, W. L., Elyria—'58
 Smith, B. A., Steubenville—'60
 Smith, J. B., Columbus—'60
 Smith, L. D., Columbus—'60
 Soderstrom, P. M., Columbus—'60
 Squire, P. D., Sandusky—'59
 Summers, M. W., Fairborn—'60
 Sumner, A. D., Cleveland Heights—'58
 Swider, D. M., Northfield Village—'60
 Thompson, J. T., Euclid—'60
 Towne, P. A., Hamilton—'58
 Upperman, M. B., Cleveland—'59
 Vegso, S. W., Dayton—'60
 Wagner, H. V., Akron—'60
 Walling, C. A., Cincinnati—'60
 Wilke, D. R., Columbus—'58
 Yunger, A. J., Toledo—'60

OKLAHOMA

Ritchey, H. C., Ardmore—'59

OREGON

Barrows, P. G., Salem—'59
 Behrens, C. A., Nehalem—'60
 Ellingson, R. S., Eugene—'59
 Ford, S. G., Portland—'58
 Hall, G. L., Portland—'58
 Heiser, J. L., Eugene—'60
 Rickard, V. L., Corvallis—'59
 Vencill, D. P., Eugene—'60

PENNSYLVANIA

Barner, J. L., East McKeesport—'60
 Bartlett, B. M., Sewickley—'58
 Bartlett, H. R., Sewickley—'58
 Bates, H., Philadelphia—'60
 Bauman, J. F., Springfield—'60

Beach, P. E., Pittsburgh—'60
 Bellak, J. L., Narberth—'60
 Bennett, D. E., New Kensington—'60
 Bonestell, W. A., Pittsburgh—'58
 Bradshaw, R. W., Philadelphia—'60
 Brill, J., Philadelphia—'60
 Brown, L. E., Philadelphia—'60
 Buller, P. P., Old Zionsville—'58
 Cardelli, D. G. E., Philadelphia—'60
 Castor, M. E., Lebanon—'60
 Catlett, E. L., Elizabethtown—'60
 Cole, E. L., Clarks Summit—'60
 Collier, H. D., Philadelphia—'60
 Crissey, R. E., Reading—'60
 Curtis, V. C., Johnstown—'60
 Demkovich, B. B., Philadelphia—'60
 Evans, M. T., Kane—'58
 Frey, D. E., Philadelphia—'58
 Gimigliano, A. T., Pittsburgh—'58
 Heine, A. L., Glenside—'60
 Idzkowsky, B. G., Pittsburgh—'60
 Jackson, B. W., Reading—'58
 Jenks, B. M., Philadelphia—'59
 Jordan, E. E., McKeesport—'60
 Kauders, D. E., Philadelphia—'60
 Keen, R. A., Pittsburgh—'58
 Kefover, M. R., Carnegie—'60
 Keisel, G. L., Meadville—'60
 Kelly, E. G., Tire Hill—'60
 Kerr, D. J., Ambridge—'60
 Kerrigan, M. E., Pittsburgh—'60
 Kimble, S. M., Lancaster—'58
 Klunen, J. A., Philadelphia—'58
 Kushner, F. J., Chester—'58
 Lane, L. E., Springfield—'60
 Larsen, M. M., DuBois—'60
 Laskin, H., Philadelphia—'60
 Libby, P. M., Fairless Hills—'60
 Likar, E. P., Meadowlands—'60
 Loar, P. J., Pittsburgh—'60
 Matrozza, F. S., Turtle Creek—'58
 McCracken, H. L., Pittsburgh—'60
 McIlvried, S. H., Philadelphia—'58
 McMillen, R. C., Beaver Falls—'61
 Mendelson, J. G., Pittsburgh—'60
 Millow, A., Scranton—'60
 Mutschler, E. M., Pittsburgh—'60
 Parks, T. B., York—'59
 Pratt, N. E., Media—'60
 Reifsnnyder, R. N., Philadelphia—'58
 Richardson, R. W., Pittsburgh—'60
 Robinson, J. W., Philadelphia—'58
 Roman, M. P., Hazleton—'56
 Scaff, B. E., Elkins Park—'58
 Schaub, J. L., Springfield—'60
 Schmidt, A. C., Allentown—'59
 Schmidt, W. M., Clifton Heights—'60
 Schnarrs, K. L., Reading—'60
 Schneiman, T. A., Philadelphia—'60
 Shaffer, M. O., Pittsburgh—'60
 Shapiro, M. P., Camp Hill—'58
 Simpson, C. C., Ambridge—'60
 Sitkoff, M., Philadelphia—'58
 Smith, C. M., Philadelphia—'58
 Smith, J. D., Ambridge—'60
 Speight, C., Greenville—'60
 Steinberg, L. H., Philadelphia—'60
 Thompson, P. H., Leetsdale—'60
 Walter, E. M., Philadelphia—'60
 Wandel, J. L., Jr., Glenshaw—'60
 Whipkey, N. G., Sewickley—'60
 Whitney, S. W., Sewickley—'60
 Wilson, M. L., Downingtown—'58
 Womack, W. F., Chester—'58
 Woodley, A. J., Philadelphia—'60
 Yalch, J. P., Bethlehem—'60
 Yatchum, J. G., Hanover—'58

RHODE ISLAND

Van Amburgh, R. B., Warwick—'58

SOUTH CAROLINA

Ashmore, K. C., Columbia—'58
 Espenshade, A. S., Greenville—'60
 Galt, L. L., Spartanburg—'60
 Williams, C. L., Greenville—'60

SOUTH DAKOTA

Allen, A. L., Sioux Falls—'60
 Russell, D. T., Hot Springs—'59
 Williams, R. R., Sioux Falls—'59

TENNESSEE

Cunningham, R. E., Jr., Bristol—'59
 Deml, W. A., Jr., Chattanooga—'60
 McKenzie, G. O., Cleveland—'58

TEXAS

Alexander, J. M., Houston—'59
 Balke, R. G., Waco—'58
 Barton, B. R., Ottine—'59
 Bethel, M. A., Houston—'59
 Bratton, S. A., Dallas—'61
 Butler, J. D., Marlin—'58
 Carrington, B. T., Cooper—'61
 Castro, J. A., Houston—'60
 Chatelain, B. M., Austin—'58
 Cicero, J. D., McKinney—'61
 Crawford, J. P., Midland—'59
 Crawford, S. T., Houston—'60
 Crisp, S. J., Corpus Christi—'60
 Cronin, J. L., Dallas—'60
 Dalton, J. C., Waco—'61
 Devine, B. T., Sheppard AFB—'60
 Duncan, E. B., Houston—'60
 Dupuy, R. J., Jr., Dallas—'60
 Ecker, R. K., Houston—'59
 Eisenberg, A. C., Ft. Worth—'60
 Ellis, A. H., McKinney—'60
 Funkey, G. G., Houston—'60
 Gage, G. A., III, Harlingen—'58
 Goodwin, J. B., Waco—'59
 Green, I., San Antonio—'46
 Hall, N. L., Premont—'61
 Harris, B. J., Cleburne—'59
 Harvill, H. C., Galveston—'60
 Hazel, S. L., Dallas—'61
 Hopkins, J. E., Houston—'59
 Hubbard, B. A., Ft. Sam Houston—'59
 James, M. J., Luling—'60
 Juday, M. A., Galveston—'60
 Kimball, R. R., Lufkin—'59
 Laird, H. L., Coleman—'61
 Langley, D. N., Big Spring—'55
 Larson, R. L., Lubbock—'60
 Lee, C. L., Dallas—'59
 Majors, N. F., Houston—'60
 McDougald, A. J., Houston—'58
 McInnis, J. A., Port Arthur—'59
 McPherson, D. W., Mesquite—'59
 Melder, F. R., Austin—'60
 Mercer, G. K., Dallas—'60
 Meyers, R. L., Abilene—'59
 Moore, E. O., Dallas—'59
 Moriarty, M. W., Dallas—'60
 Mosallem, N. J., San Antonio—'60
 Nash, J. T., La Marque—'60
 Nunnery, B. L., Dallas—'61
 Oxford, P. R., Dallas—'61
 Palmer, B. J., Houston—'58
 Parker, A., Dallas—'60
 Pavlis, P. M., Ft. Sam Houston—'59
 Poynor, R. N., Dallas—'60
 Robinson, T. M., Richardson—'59

Taggart, A., Luling—'60
 Thompson, M. J., Lubbock—'59
 Turner, G. A., Jr., Gonzales—'59
 Walters, M. N., Pasadena—'59
 Wendeborn, J. D., Sagerton—'60
 Weston, M. J., Dallas—'60
 Womack, P. A., Ft. Worth—'59
 Wyatt, D. L., San Antonio—'60
 Wythe, G., Jr., Temple—'58

UTAH

Durham, B. C., Logan—'59
 Noble, B. J., Salt Lake City—'58
 Slade, T. E., Salt Lake City—'60

VIRGINIA

Bailey, S. A., Richmond—'59
 Fontana, H. A., Jr., Alexandria—'58
 Hayman, J. W., Warrenton—'60
 Hubbard, P. T., Richmond—'58
 Kimble, L. M., Fairfax—'58
 Kreider, R. R., Richmond—'58
 McDonald, M. E., Richmond—'58
 Nesselthaler, J. V., Fredericksburg—'60
 Nuttycombe, E. J., Richmond—'59
 Odenkirk, B. J., Arlington—'59
 Patterson, C. N., Roanoke—'53
 Sheffer, C. L., Jr., Richmond—'58
 Showalter, J. C., Richmond—'58
 Spala, H. M., Ft. Monroe—'59
 Spencer, M. M., Richmond—'59
 Stewart, E. P., Richmond—'58
 Townsend, R. E., Danville—'58
 Tullos, R. G., Hampton—'59
 Warner, S. S., Norfolk—'59
 Wiseman, N. L., Arlington—'60
 Yurko, L. M., Richmond—'60

WASHINGTON

Brooks, B. J., Seattle—'60
 Dammeier, A. A., Big Harbor—'60
 Davis, E. P., Seattle—'60
 Dublinski, L. H., Walla Walla—'60
 Eshleman, D. M., Seattle—'60
 Inman, D. A., Seattle—'60
 Johnsen, F. E., Seattle—'59
 Kalisiak, L., Seattle—'60
 Keogh, R. B., Tacoma—'53
 King, P., Tacoma—'43
 Laffaw, S. A., Seattle—'59
 Lewis, D. W., Wenatchee—'60
 Mills, S. V., Seattle—'60
 Milner, R. N., Cashmere—'60
 Nixon, M. G., Seattle—'41
 Rahe, L. D., Seattle—'60
 Reid, B. B., Bellevue—'60
 Robbers, D. L., Pullman—'58
 Robinson, C. P., Seattle—'59
 Sebastian, S. L., Seattle—'59
 Van Divort, J. P., Seattle—'60
 Van Patten, E. L., Walla Walla—'60

WEST VIRGINIA

Cook, M. B., Williamson—'58
 Dillon, H. L., Institute—'60
 Gouzd, R., Sr., Fairmont—'60
 Hightower, A. B., Beckley—'60

Moran, R. A., Charleston—'60
 Scott, J. L., Beckley—'59
 Skos, F. M., Wheeling—'60
 Vespaziano, J. L., Morgantown—'60
 Zimmerman, J. E., Beckley—'58

WISCONSIN

Cammack, J. M., Milwaukee—'60
 Carlson, J. A., Oshkosh—'60
 Faber, M. A., Milwaukee—'58
 Hupprich, E. M., Madison—'59
 Jones, D. C., Appleton—'59
 Larson, R. D., Viroqua—'59
 Laubach, B. M., Milwaukee—'59
 Natrop, C. M., LaCrosse—'59
 Oldenburg, D. J., LaCrosse—'60
 Osterberg, R. D., Florence—'59
 Peik, N. L., Sheboygan—'60
 Petrykowski, C. A., Hales Corners—'59
 Siegel, M. R., Milwaukee—'61
 Taugher, B. D., Oshkosh—'58
 Varney, M. J., Milwaukee—'59
 Waldschmidt, J. C., Milwaukee—'59
 Westenber, B. P., Cambria—'58
 Zupan, Sr. M. D., Milwaukee—'59

WYOMING

Barnes, R. L., Cheyenne—'60
 Chadwick, D. B., Kemmerer—'53
 Larkin, L. H., Sheridan—'60
 Roberts, P. R., Cheyenne—'60

PUERTO RICO

Christian, M. d. P., Mayaguez—'60
 de Rodriguez, C. C., Santurce—'52
 Santana, E. R., Cabo Rojo—'60
 Villanueva, E. E., Ciales—'60

FOREIGN

Frickson, L. M., Assam, India—'60
 Ladion, H. d. G., Manila, Philippines
 Vazquez, A. J., Mendoza,
 Argentina, S. A.—'60

ADDRESS UNKNOWN

Brody, M. K.,—'58
 Gardner, M. C.,—'60
 Gault, C. R.,—'58
 Glover, D. K.,—'43
 Graham, G. M.,—'60
 House, A. M. D., Jr.,—'59
 Huggett, P. G.,—'59
 Klatt, P. R.,—'48
 Mason, P. B.,—'58
 Romine, M. G.,—'60
 Roof, M. S.,—'58
 Sander, D. Y.,—'58
 Shoemaker, A. L.,—'58
 Skinner, R. D.,—'59
 Smith, A. J.,—'60
 Snee, J. F.,—'58
 South, B. D.,—'59
 Thompson, J. S.,—'58
 Troske, F. H., Jr.,—'59
 Ulrich, F. T.,—'58
 Weiland, L. H.,—'58



Deceased Members

DR. GUSTA DAVIDSOHN

Gusta Davidsohn of Chicago died on December 24, 1960. Dr. Davidsohn was born in Tarnopol, Austria on June 30, 1902. She received her medical education at the Universities of Vienna and Warsaw, took her residency at Childrens Hospital in Berlin, Germany and served an internship at Lakeview Hospital in Chicago. She was attending physician in Physical Medicine at Mount Sinai Hospital in Chicago since 1941 and Director of Physical Medicine and Rehabilitation, as well as Associate Professor of Medicine at the Chicago Medical School since 1949. Dr. Davidsohn was also past president of the Chicago Society of Physical Medicine and Rehabilitation.

DR. GEORGE E. HAYWARD

George E. Hayward, Meadville, Pa., passed away on March 22, 1960. He was graduated from Hahnemann Medical College and Hospital of Philadelphia and licensed to practice medicine in Pennsylvania in 1910. Formerly Mayor of the city, he was for many years a member of the Board of Health of Meadville and served as chairman of the Meadville Civil Service Commission. Dr. Hayward was currently associated with the Meadville City Hospital where he died at the age of 74 of a cerebral hemorrhage.

DR. STANLEY HORN

Stanley Horn of Canada died suddenly at Miami, Florida on January 28, 1961 at the age of 70. He was born in Warsaw, Poland and received his medical education in France at the Universities of Montpellier and the Sorbonne. In 1928 he came to the United States and set up a practice in New York City. He was Chief of Staff in the Department of Physiotherapy at the Bronx Hospital, N. Y. Dr. Horn moved to Sherbrooke, Quebec in 1938 and established the Medical Electrosurgical Clinic on Court Street where he practiced until the time of his death.

DR. ARCHIBALD P. HUDGINS

Archibald P. Hudgins, Charleston, W. Va., passed away October 27, 1960 at the age of 58 of a cerebral hemorrhage. He was graduated from the Medical College of Virginia in 1928. At one time, he served at Hinton, W. Va. Hospital; was affiliated with Kanawha Valley, McMillan, and Memorial Hospitals; and formerly held office as secretary of the Summers County Medical Society. Dr. Hudgins was also a fellow of the International College of Surgeons.

DR. WILLIAM H. NORTHWAY

William H. Northway of Palo Alto, California had been in ill health four months prior to his death. He passed away on December 23, 1960 at the age of 57 after a heart attack at his Palo Alto home. He was born April 23, 1903 in Nunda, N. Y. He was graduated from Stanford University in San Francisco in 1930. Dr. Northway was Professor and Medical Director of Physical Medicine at Stanford. In 1947 he was appointed Assistant Dean of the School of Medicine. His society memberships include the American Rheumatism Association.

IF YOU ARE

- a clinician
- a research worker
- a physiologist
- a bio-physicist
- a teacher of physical medicine and rehabilitation

IF YOU WANT

- to stay abreast with what is new in physical medicine and rehabilitation

YOU MUST

- read the **Archives of Physical Medicine and Rehabilitation**, the official journal of the American Congress of Physical Medicine and Rehabilitation and the American Academy of Physical Medicine and Rehabilitation
- no other medical periodical gives you as broad a coverage in the field of physical medicine and rehabilitation . . . each month you will find in this journal informative articles on new developments, theories and practices dealing with all phases of this specialty

YOU ARE INVITED

- to send in your subscription today. Sample copy will be sent on request. Subscription price \$8.50 per year; Canada, \$9.50; elsewhere, \$14.00 the year. Bill later if you wish. Return the coupon with your instructions.

AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION

30 No. Michigan Ave., Chicago 2.

Please find enclosed check for \$_____ ☐ or bill me ☐ for one year's subscription to the ARCHIVES.

Name _____

Address _____

City, Zone and State _____

American Academy of Physical Medicine and Rehabilitation

Officers and Committees

1961



Officers

President — Ray Piaskoski
President-Elect — Robert W. Boyle
Vice-President — Max K. Newman
Secretary — Harriet E. Gillette
Treasurer — James W. Rae, Jr.
Executive Secretary — Dorothea C. Augustin

Equipment and Facilities in Physical Medicine and Rehabilitation

Ralph E. Worden, *Chairman*
Robert L. Bennett
William J. LaJoie
James W. Rae, Jr.
Joseph W. Schaeffer
Edwin C. Welsh

Board of Governors

Robert W. Boyle
Clarence W. Dail
Harriet E. Gillette
Morton Hoberman
A.B.C. Knudson
Max K. Newman
Ray Piaskoski
James W. Rae, Jr.
Herman L. Rudolph

Medical Practice in Physical Medicine and Rehabilitation

Max K. Newman, *Chairman*
Lee B. Greene
Arthur C. Jones
Miland E. Knapp
Leo Rosenberg
Oscar O. Selke, Jr.

Advisory Committee for Advancement of Physical Medicine and Rehabilitation

Clarence W. Dail, *Chairman*
Frances Baker
Joseph G. Benton
Ralph E. De Forest
Earl C. Elkins
Donald L. Rose
Charles D. Shields

Membership

Lewis A. Leavitt, *Chairman*
Bernard J. Michela
Arthur A. Rodriguez

Nominating

Edward W. Lowman, *Chairman*
H. Worley Kendell
Frank H. Krusen

By-Laws

Shelby G. Gamble, *Chairman*
Gordon M. Martin
Rex O. McMorris
Herman L. Rudolph
Jerome S. Tobis
Reuben Wasserman

Scientific Program

Miland E. Knapp, *Chairman*
Robert C. Darling
George C. Twombly, Jr.

Members

Standing Committees of the

American Congress of Physical Medicine and Rehabilitation



1961

Awards and Prizes

Jerome S. Tobis, *Chairman*
Robert C. Darling
William J. Erdman, II
Murray M. Freed
Harriet E. Gillette
Ernest W. Johnson
Lewis A. Leavitt
Milton Lowenthal
Louis B. Newman
Charles D. Shields

Constitution and By-Laws

Robert C. Darling, *Chairman*
Michael Kosiak
John H. Kuitert
F. Manley Brist, *Ex-officio*

Legislation

G. Keith Stillwell, *Chairman*
Henry Fleck
Bernard J. Michela
Oscar O. Selke, Jr.
Charles D. Shields
Walter J. Treanor
Herman Wing
F. Manley Brist, *Ex-officio*

Medical Practice

Lee B. Greene, *Chairman*
Herman J. Bearzy
Miland E. Knapp
Edward M. Krusen, Jr.
William J. LaJoie
Arthur A. Rodriguez
Leon M. Rothman

Membership

Herman L. Rudolph, *Chairman*
John W. Goldschmidt
Bruce B. Grynbaum
A.B.C. Knudson
Elizabeth S. Austin

Nominating

Frederic J. Kottke, *Chairman*
A.B.C. Knudson
Edward W. Lowman
Fred B. Moor
Harold N. Neu

Professional Education and Development

Justus F. Lehmann, *Chairman*
Arthur S. Abramson
Joseph G. Benton
Michael M. Dacso
Ernest W. Johnson
Lewis A. Leavitt
Charles Long, II
Donald L. Rose

Program

Eugene Moskowitz, *Chairman*
Charles O. Bechtol
Gustave Gingras
Gordon M. Martin
William A. Spencer
Frederick E. Vultee, Jr.
Glenn Gullickson, Jr., *Ex-officio*

Public Relations

Jerome S. Tobis, *Chairman*
Lewis A. Leavitt
Charles D. Shields

Specialized Hospitals and Services

Frederick E. Vultee, Jr., *Chairman*
Arthur S. Abramson
Robert L. Bennett
Michael M. Dacso
Charles H. Flint
Morton Hoberman
James W. Rae, Jr.
Jack Sokolow
Dorothy M. Stillwell

CONTENTS



Recognition and Care of Early Scoliosis. Robert L. Bennett, M.D.	211
◇	
Mechanical Properties and Temperature of Intact Skeletal Muscle in Patients with Muscular Dystrophy. Stella Y. Botelho, M.D.; Eleanor Bendler, M.D., and Sibyl B. Beckett, M.S.	226
◇	
Analysis of Sounds from Normal and Pathologic Knee Joints. Herbert Fischer, M.D., and Ernest W. Johnson, M.D.	233
◇	
Effect of Short Wave Diathermy on Radio-Sodium Clearance from the Knee Joint in the Normal and in Rheumatoid Arthritis. Dr. Ronald Harris.	241
◇	
Evaluation of Physical Disabilities by Means of Patient Profile Chart. O. Leonard Huddleston, M.D.; Richard W. Moore, M.D.; David Rubin, M.D.; Thomas L. Humphrey, B.S., R.P.T.; John W. Campbell, B.S., O.T.R., and Ron Blanchette.	250
◇	
Application of the Stretch and Hoffman Reflexes to the Objective Measurement of Spasticity. Osvaldo E. Miglietta, M.D., and Milton Lowenthal, M.D.	258
◇	
Special Rehabilitation Services in Certain Cardiac and Pulmonary Disabilities. Charles D. Shields, M.D., and Margaret M. Kenrick, M.D.	265
◇	
Cancer Arthritis and Rheumatoid Arthritis. Dr. Brynjulf Strandberg, and Dr. Niels V. Jarlov.	273
◇	
Role of Orthotics in Rehabilitation of Hands in Quadriplegia of Spinal Origin. Odon F. von Werssowetz, M.D.	279
◇	
Use of Nylon "Muscle" in Functional Bracing in Severe Quadriplegia. John S. Young, M.D.; Bruce Scott, C.O., C.P.; Virginia Gordon, O.T.R., and Elnora Gilfoyle, O.T.R.	286
◇	
White House Conference on Aging: Policy Statement on Rehabilitation.	290
◇	
Supplement S-1 — 14th Edition Directory, American Registry of Physical Therapists.	293



